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...OF TRAINED AND UNTRAINED RATS....

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THE EFFECT OF PROLONGED USE OF ANABOLIC STEROIDS ON ENERGY SOURCES OF TRAINED AND NONTRAINED RATS

by

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A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH

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DEPARTMENT OF PHYSICAL EDUCATION

EDMONTON, ALBERTA

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FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled "The Effect of Prolonged Use of Anabolic Steroids On Energy Sources of Trained and Nontrained Rats" submitted by William Charles Fielding in partial fulfilment of the requirements for the degree of Master of Science.

DEDICATION

To Mr. J. Stanley Hill, Assistant Professor,
Faculty of Physical Education, University of Western Ontario,
for his friendship and inspiration.

ABSTRACT

Fifty-four albino rats (Wistar strain) were used to determine the effect of prolonged use of anabolic steroids on energy sources of trained and nontrained rats.

Animals were divided into two equal groups: one group was bilaterally castrated while the second served as non-castrated controls. One half of each group received Dianabol (Methandrostenolone - 17α - methyl - 17β - hydroxy-androsta - 1, 4 - dien - 3 - one) orally administered weekly (1.75 mg/kg). Two thirds of each group were trained to exercise on a motor driven rodent treadmill by progressively increasing the speed and duration of the daily exercise session until they were capable of running continuously for one hour at a speed of 1 mph. Each animal then continued to run at this intensity for an additional 8-9 weeks. One half of the trained animals were sacrificed immediately after a fatiguing run while all other animals were sacrificed at rest.

Analysis of the results indicated that regular exercise had no effect on the parameters measured with the exception of adrenal weights which increased slightly as a result of training. When the animals were exercised to exhaustion decreases occurred in liver and spleen weights, skeletal muscle and liver glycogen, and blood glucose levels. Increases were apparent in adrenal weight, blood lactate,

plasma FFA and the mobilization of lipid FFA. Analysis also revealed that castration caused decreases in body and liver weights as well as increases in blood lactate levels.

Anabolic steroid treatment did not affect body weight, testicular size, spleen, heart, adrenal weights, blood lactate, resting blood glucose levels or resting FFA levels. Decreases, however, were noted in liver weights and resting skeletal muscle glycogen levels. When the animals were exercised to exhaustion it was apparent that anabolic steroids had no effect on the depletion of glycogen stores or on the utilization of blood glucose. Although mobilization of FFA from adipose tissue occurred during exhaustive exercise in normal rats, the anabolic steroid treated animals were not able to mobilize FFA as readily.

Within the limitations of the study it was concluded that anabolic steroids had a significant effect on skeletal muscle glycogen sources and the mobilization of FFA sources with exercise and training.

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STATEMENT OF THE PROBLEM

Introduction

In recent years the non-medical use of anabolic steroids has been a subject of much controversy in the athletic world. With so much emphasis being placed on winning and excellence, coaches and athletes have constantly been searching for artificial means to improve performance particularly in the area of "ergogenic aids". These ergogenic aids can function in one of two ways: (a) to improve the capacity of the muscles to do work, or (b) to remove or reduce inhibitory mechanisms in order to enhance performance.

The chief function of anabolic steroids is to stimulate the synthesis of cellular protein (129). Anabolic agents have been used for some time for patients recovering from illness or after surgery, for treatment of osteoporosis, fracture healing, severe burns, muscular dystrophy, for protein tissue building, and for myotrophism. Anabolic agents have also been credited with having an important effect in stimulating the appetite and imparting a feeling of well being (4).

In the past the use of anabolic steroids was largely limited to athletes participating in events in which success

required great muscular mass and strength. At the present time these drugs appear to be more widespread among athletes in various other sports. This use of anabolic steroids by athletes has received sharp criticism by the medical profession (155) and a number of sport federations (60, 153).

Anabolic steroids are derivatives of the male sex hormone testosterone. Chemical modifications of testosterone have led to some synthetic compounds which show a satisfactory dissociation between anabolic and androgenic properties. Many attempts have also been made to find orally active anabolic steroids without androgenic activity since orally active compounds would have a much greater clinical application than intramuscularly administered drugs. Derivatives synthesized in recent years often feature alkyl and/or electronegative halogen substituents. Numerous derivatives of anabolic steroids have been prepared for oral administration by 17α- alkylation. Alkylation in the 17α- position appears to be a factor blocking the dehydrogenation of the 17β - hydroxyl group. The 17α - alkyl testosterone derivatives owe their oral activity to the protection of the 17β - hydroxyl group. Methandrostenolone, an orally active anabolic steroid, is a 17α - alkyl substituted compound showing strong anabolic properties (43, 44).

Statement of the Problem

The purpose of this investigation was to examine the effect of an anabolic steroid, castration, and fatigue on energy sources of trained and nontrained rats. The following parameters were measured: total body weight, organ weights, muscle and liver glycogen, blood glucose, blood lactate, plasma free fatty acid (FFA) levels and lipid FFA mobilization.

Rationale Behind the Study

Previous studies on anabolic steroids have primarily been carried out in a clinical situation. Kochakian et al (120-126), Korner and Young (128) and Desaulles (43) examined the myotropic action of androgens and several anabolic steroids on intact, castrated, adrenalectomized or hypophysectomized rats and guinea pigs. It was found that steroid dosage and species had a profound influence on the character and localization of the myotropic effect. Studies investigating body and organ weights are conflicting (51, 110, 150). Methandrostenolone had a hypoglycemic effect on fasting subjects, while no effect on blood pyruvate levels were noted.

Those investigators examining the effects of anabolic steroids on athletes have dealt with the areas of muscular strength (110, 111, 178), power (111) and oxygen uptake (55, 111, 178). A few reports examining blood chemistry profiles were also noted (98, 155).

In spite of the widespread use of anabolic steroids there is little understanding of their mechanism of action. Investigations examining the effect of anabolic steroids on sources of energy mobilization are very limited (191). Therefore, a study was necessary to examine selected variables in the mobilization of energy.

Limitations and Delimitations of the Study

1. The study was confined to the male Wistar rat between ages seven to 24 weeks.

2. Food intake was not controlled.

3. Only one intensity of endurance training was used, and some trauma could have resulted from the presence of the electrical shocking device on the treadmill.

4. Differences in body weights were not accounted for when analyzing organ weights.

CHAPTER II

REVIEW OF LITERATURE

Anabolic Steroids

Anabolic steroids may be defined as those steroids whose main function is to generally stimulate the synthesis of cellular protein (129). Anabolic agents are usually derivatives of testosterone and show a relatively stronger anabolic than virilizing effect.

Testosterone and its derivatives have long been used for their anabolic effects, however, their usefulness has been limited because of their androgenic properties. Attempts to dissociate the anabolic effect from the androgenic effect by modification of the testosterone molecule have resulted in the development of several synthetic steroids. Laboratory tests and animal assays indicate that a wide separation of anabolic and androgenic effects has been achieved, although the nitrogen-retaining and androgenic potencies of these compounds in human beings cannot be predicted accurately from these tests (4).

An important class of androgenic and anabolic compounds was synthesized with the direct purpose of avoiding metabolic inactivation. Introduction of a 17α - alkyl substituent into the testosterone molecule changes the secondary 17β - hydroxyl group to a tertiary hydroxyl group. Thus metabolic inactivation through oxidation of

CHAPTER II

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the 17β - hydroxyl group to a 17 - keto group is no longer possible. The 17α - alkyl testosterone derivatives owe their oral activity to the protection of the 17β - hydroxyl group. The presence of the alkyl group is responsible for the undesirable side effects including liver tissue damage (202).

Methandrostenolone, 17α - methyl - 17β - hydroxyandrosta -1, 4 - dien - 3 - one ($C_{20}H_{28}O_2$), is a 17α - alkyl substituted steroid. It is a synthetic steroid hormone, both structurally and pharmacologically related to other androgenic anabolic steroids. The preparation of this compound was a result of investigations on the effect of additional unsaturation in testosterone derivatives. Two methods have been worked out for the synthesis of methandrostenolone (200, 201): (a) the action of the fungus Didymella on 17α - methyltestosterone (microbiologic dehydrogenation) and (b) chemical dehydrogenation of 17α methyltestosterone with selenium dioxide, tertiary butanol, and acetic acid. Methandrostenolone has been found to show strong anabolic properties and low androgenic activity (43, 44, 202). Typical of androgens, methandrostenolone causes retention of nitrogen in the form of protein and of calcium, sodium, potassium, chloride and phosphate, leading to an increase in skeletal weight (5, 143).

Some direct connection between the muscle mass of the body and the activity of androgens has been suspected for a long time. Extensive investigations by Kochakian et al

(120-126) yielded important results with respect to the myotropic action of androgens and several anabolic steroids. Castration of growing male rats and guinea pigs results in a gradual slowing down of the increase in weight. In the rat, all skeletal muscles and the skin participate in this change in proportion (126), whereas, in the guinea pig, some muscles are affected more strongly by this involution than others (124). The involution of muscles after castration can be reversed by the administration of androgen (120, 124); however, only very massive dosages of androgen are able to effect a gain in weight above normal values (121).

Similar investigations by Korner and Young (128) on intact, adrenalectomized, or hypophysectomized rats showed that the additional factors of age, sex, and steroid dosage have a profound influence on the character and localization of the myotropic effect.

Desaulles (43) reported that castrated rats as well as adrenalectomized and hypophysectomized animals appeared to be more sensitive than normal animals when treated with methandrostenolone. These results demonstrate that the action of methandrostenolone on weight gain is an intrinsic component of its properties. The same effect on normal animals, however, still depends on the presence of a functioning pituitary.

A few contradictory reports exist concerning changes

of the chemical composition of muscle after castration, substitution of androgen, or after treatment with anabolic steroids. In the guinea pig, castration resulted in the decrease in protein, water, and ash content in proportion to the decrease in the weight loss of the muscle. With androgen support, all these changes were reversed (122, 125). The myoglobin content of the muscle, however, was independent of the androgen level (175), and after castration and subsequent androgen replacement there were no changes in concentration of other protein fractions of the muscles, such as myosin, sarcoplasm, and collagen (174).

Total Body and Organ Weights

A number of investigators have conducted research to determine the physiological function of onternal organs during exercise (8, 9, 15, 40, 49, 82, 90). Price-Jones (158) investigated the effect of exercise on several physiological and anatomical parameters in young rats. It was found that the rate of growth (in terms of body weight) was 1.25 to 1.44 times as great in the resting rats as compared to the active animals. Similar results by other investigators have also been reported with regard to body weight (40, 52, 88, 182). In the fifth part of a series of experiments by Stevenson et al (185) it was found that exercised animals lost weight, the amount of loss being approximately proportional to the amount of exercise. McClintock et al (144) and others (49, 193) re-

ported that exercise produced no change in total body weight.

A study by Hatai (82) in 1915 on albino rats reported that exercised animals had heavier heart weights than controls. The same results were noted for kidney, testicles, and liver weights. Spleen weights, however, were larger in the control animals. Steinhaus et al (182) noted similar results with the exception of the testes which decreased in weight with exercise. Several other researchers recorded similar results in determining the effect of exercise on total body and organ weights (40, 49, 88). Montoye et al (148) found no significant difference in organ weights between exercised or control rats. McClintock et al (144) as well as Tepperman and Pearlman (193) recorded similar results for heart weights in rats.

In examining the effects of exercise on spleen weights a significant loss in weight was noted following fatigue (15, 74, 194). Although this weight loss was once attributed to expulsion of the stored blood from the spleens (15, 182), others (73, 74) do not agree with this phenomena as similar results were reported in animals not exercised to fatigue.

Relatively few studies have been conducted to determine the effect of exercise and anabolic steroids on organ weights. Johnson et al (110) in a study on students aged 18 to 38 years found that anabolic steroid treatment resulted in a significant weight gain over the control or

placebo groups. Eagen et al (51) and Murphy et al (150) noted that heart weight was greater in the steroid group but not in the exercise-steroid group where a myotropic increase may have been balanced by a fat content decrease. The pituitary gland and testes size was greater in the exercise group but depressed in the steroid group. Treatment with testosterone proprionate and other anabolic steroids have been demonstrated to produce hypertrophy of atrophied and normal musculature (43).

Muscle Glycogen and Blood Glucose

Carbohydrates are stored in the form of glycogen in the skeletal muscle and liver tissue until such time as it is needed as glucose to provide energy for muscular work (81, 195). The utilization of glycogen in the muscles is dependent upon a complex series of enzymatic reactions which is initiated by release of adrenaline from the adrenal medulla (181). The release of adrenaline is propagated by stimulation from the nervous system in response to severe and sudden muscular exertion. All of these events result in a rapid breakdown of glycogen to glucose-1-phosphate. When breakdown is in progress, the enzymes for resynthesis is proceeding, the enzymes involved in catabolism are inhibited. The preceding reactions take place in the skeletal muscles, but similar reactions also take place in the liver, to maintain glucose levels in the blood (145).

A normal level of blood glucose of about 70 to 90 mg% is always present in the blood and is available for immediate use (208). Blood glucose levels are maintained by homeostatic mechanisms which are hormonal devices for controlling the rates of glycogenesis, glyconeolysis and glyconeogenesis and glucose metabolism by all cells (7).

In moderate exercise, which is essentially aerobic and of relatively short duration, glucose levels change very little. Hermansen et al (91) reported that in initial tests of intermittent exercise, glucose levels did not change. Robinson (166) found that for all age groups 6 to 75 years the glucose levels rose only about 10 mg% over resting levels. Ahlborg (1) showed that in the initial stages of his prolonged work experiments glucose levels rose only slightly.

In maximal work, when the subject is exhausted in a relatively short time, significant increases in glucose and insulin concentrations have been reported. Robinson (166) noted a state of hyperglycemia after a short maximal effort for all age groups. The highest values appeared between the ages of 13 to 29. Hermansen et al (91) reported an increase from 79.2 mg% at rest to 172.3 mg% after the last bout of heavy intermittent exercise. Plasma insulin also increased greatly, the peak levels occurring approximately ten minutes after the peak values for glucose.

If exercise continues for a prolonged period of time, the glycogen stores can be greatly depleted. At the same

time that glycogen is being utilized, there is an increased utilization of glucose coming from the liver so that at the end of the exercise period blood glucose levels are also low (12). A decrease in blood glucose levels after prolonged work has also been reported by other investigators (1, 115, 134).

In a series of experiments Hultman (100) reported that the average glycogen content in the quadriceps femoris muscle of humans was 1.51 gms/100 gms using the Diazyme-glucose oxidase method and 1.31 gms/100 gms using the TCA-toluidine method. Prolonged exercise to exhaustion produced values of 0.41 gms/100 gms and 0.27 gms/100 gms respectively.

During training the glycogen content of certain muscles increases during the early weeks of training in dogs (159). This increased glycogen content of skeletal muscles does not appear to be essential for the increased efficiency which results from training. It has also been reported that the skeletal muscle glycogen increased when rats were trained to run (205). When the animals were exercised to exhaustion the glycogen levels in skeletal muscle and liver tissue was depleted. This loss of glycogen from the liver and skeletal muscle during the exhaustive run has been interpreted as meaning that the glycogen is being utilized to provide energy for the performance of the work. An increase of glycogen in cardiac tissue has also been noted after training (50). The evidence seems conclusive that the trained animals have higher glycogen levels in both

liver and muscle than do nontrained animals and they do not deplete their glycogen levels as quickly.

Literature reviewed for this experiment revealed that few studies have investigated the effect of exercise and anabolic steroids on glycogen or blood glucose levels. Landon et al (130, 131, 132) studied the effects of methandrostenolone on carbohydrate metabolism and blood sugar levels in clinical situations. In the majority of subjects, methandienone lowered the fasting blood sugar level and impaired tolerance to both orally and intravenously administered glucose. The hypoglycemic effect of methandrostenolone was related to dose. Methandrostenolone appeared to have no effect on blood pyruvate levels in fasted subjects nor on pyruvate curves following oral and intravenous glucose. Anabolic steroid treatment also decreased the hyperglycemic effect of glucagon, and the associated changes of the plasma inorganic phosphorous were less than normal. The hyperglycemic effect of adrenalin and the accompanying changes in plasma inorganic phosphorous levels were apparently unaffected by methandrostenolone administration.

Talaat et al (186) in examining the effects of testosterone proprionate on blood sugar and on the composition of the liver and muscles of male rabbits reported a significant increase in liver weight and in liver glycogen using a daily dose of 1 mg/kg for 10-20 days. A daily dose of

5 mg/kg not only resulted in the same effect on the liver but also increased muscle glycogen content and blood sugar level. Higher doses led to the opposite results. Similar results were reported in a later study by Talaat et al (187).

Taylor et al (191) found that anabolic steroids had no effect on the resting levels of heart or skeletal muscle glycogen levels in rats. It was also noted that the mobilization of glycogen was not affected.

Blood Lactate

In most instances it is implied that the efficiency of the various pathways and consequently the production of energy is related to the availability of oxygen to the metabolizing system. If the oxygen is readily available the system functions to optimum capacity with little metabolite accumulation. If there is an inadequate supply of oxygen however, as during altitude exposure or anemia, certain cardio-respiratory ailments, or exercise, the capacity of the system to produce energy is reduced and the subsequent production of intermediate metabolites is increased (138). The most abundant metabolite of anaerobic muscular metabolism is lactate. Owing to relative anoxia of the muscles, lactate accumulates therein and diffuses into the blood stream (116).

It had early been realized that lactate production during muscular contraction was formed from the glycogen stored in the muscle and that these reactions formed the

major energy source during anaerobic contractions (69, 171). With the conversion of glucose or glycogen to lactate, a limited amount of energy in the form of ATP is provided when the oxygen supply is deficient. This anaerobic pathway for energy production thus enables a tissue to maintain it's function to a certain degree despite hypoxic conditions (77, 208).

The anaerobic oxidation of glycogen or glucose is made possible by the simultaneous reduction of a coenzyme nicotinamide adenine dinucleotide (NAD) which acts as a hydrogen acceptor. The formation of lactate resulting from the reduction of pyruvate, thus enables the $NADH_2$ to be reoxidized allowing further glycolysis to take place. The formation of lactate, although not necessary for the delivery of energy, serves as a storehouse for hydrogen ions and thereby keeps the reaction continuing (12). Under anaerobic conditions the accumulation of lactate or the exhaustion of the supply of glycogen or glucose may limit the cell activity.

Normal resting levels of lactate for humans range from 8.0 to 20.0 mg% (34, 142, 167, 208). Since the demonstration by Ryffel (171) of an increased lactate concentration in the blood and urine of man after muscular activity, a number of reports have appeared on the general subject of lactate in relation to exercise (93, 99, 142). Hill (92), Margaria (142) and Huckabee (99) found that blood lactate of men

following heavy stress varied with oxygen debt. From these and other results it is now possible to represent changes in blood and urine lactate during and following muscular activity.

In moderate exercise, the oxygen supply to the muscles is sufficient to completely oxidize the pyruvate. Initially the lactate level may rise slightly until a steady state is reached after which the concentration will return to near resting values (119, 142, 206). However, as the level of exercise stress increases there is a greater breakdown of glycogen and the reduction of NAD is speeded up. If oxygen cannot be supplied in sufficient quantity to the increased demand, the pyruvate acts as a hydrogen acceptor forming lactate in greater quantity. It is apparent that lactate appears in excess at about 40 to 50% of maximal oxygen uptake in the nontrained subject and about at 60 to 70% in the trained individual (12, 119, 142).

Various researchers have reported peak lactate levels for well trained athletes. Astrand et al (10) reported a peak of 142.0 mg% for one male athlete. A range of 160.0 mg% to 175 mg% was noted for four male athletes by Astrand and Saltin (13). Karlsson et al (114) reported a mean value of 141.0 mg% for a group of male and female athletes.

A review of the literature revealed that no studies had investigated the effects of anabolic steroids on blood lactate.

Free Fatty Acids

Muscular work, performed aerobically in the post-absorptive state, depends mainly on the utilization of fat (46, 61). It has only been in recent years that considerable research has been conducted on the role that fat, in the form of free fatty acids (FFA), plays in energy metabolism (29, 30, 57, 58, 62). Interest in this area developed when it was noted that FFA, although contributing only 5% of the total lipids in the plasma, comprised a fraction which responded consistently to a variety of physiological stimuli (45, 75).

There are three main, functionally different, fatty acid transporting classes in the blood lipid transport system. The first class is the chylomicrons which transport the dietary fatty acids after absorption from the gastrointestinal tract. The fatty acids transported from the liver to the other tissues are contained in the triglycerides of the lipoproteins, which constitute the second major transport class. The third class is FFA; the form in which fat is continuously mobilized from adipose tissue. It is presently believed that a free fatty acid-albumin complex is the most important system for transport of endogenous fatty acids from the storage sites for utilization by the muscle tissue (18, 61).

It has generally been observed that resting levels of FFA in man are in the range 0.30 to 0.60 uEq/ml. During

submaximal exercise two characteristic changes occur in plasma FFA levels. An initial fall and a subsequent rise have been studied and their mechanisms partly elucidated. The early fall in FFA is ascribed to increased perfusion of muscle tissue with blood during exercise (29, 30). The late rise is related to increased activity of the sympathetic nervous system (59, 85, 86, 105, 156), growth hormone (102, 163) and thyroid stimulating hormone (199). This elevation in plasma and adipose tissue FFA following submaximal exercise indicates that lipid mobilization in the fat depots is stimulated by muscular work (18, 30, 63, 68, 86, 105, 112).

Maximal exercise, however, causes a decrease in plasma FFA concentration (18, 30, 35, 109, 168). This decrease has been attributed to both an increase in uptake by the working muscle and a decreased blood supply to the fat pads. It has also been found that in the presence of lactate, glucose, and insulin the production of FFA from the adipose tissue will be decreased (54, 160, 161, 170, 207). There are some indications that fatty acids may be furnished to muscles from lipid pools within the muscle itself (27, 31).

Literature reviewed for this study revealed that very few researchers have investigated the effect of anabolic steroids on free fatty acid levels.

CHAPTER III

METHODS AND PROCEDURES

Fifty-four albino male rats of the Wistar strain with initial body weights ranging from 180 grams to 210 grams were used in this study. Each animal was housed in an individual 7 x 10 x 7 inch self-cleaning cage in a laboratory controlled automatically for room temperature (24 $\pm$ 1^{o}C) and 12 hour light-dark cycle (8 a.m. - 8 p.m.).

Eighteen animals were placed in a nonexercise control group while 36 animals were trained to run on a motor-driven treadmill (Quinton Rodent Treadmill or Collins Treadmill). The speed and duration of the daily exercise sessions were progressively increased until the animals were able to run continuously for one hour at one mile per hour, five days per week, at the end of five weeks. Each animal then continued to run at this intensity for an additional 8-9 weeks. Mild electrical stimulation (140 mv maximum) was used initially to teach the rats to run. The control animals received only the exercise natural for nutrition and excretion. Half of the animals in each group were castrated at approximately 7 weeks of age in order to eliminate the major supply of testosterone (a natural androgen) to the body.

Methandrostenolone (Danabol co-donated by CIBA-GEIGY CANADA LIMITED) was orally administered to half of the

animals in each group at a dosage of 0.25 milligrams per kilogram body weight per day given on a weekly basis. At the beginning of each training week the methandrostenolone dosage prescribed for each rat was mixed with approximately 35 mgs of powdered food consumed by the animals. A plaster vial containing the powdered food and drug was then attached to the inside of each cage; in front of an individual feeder. To insure the correct dosage the solid food pellets, contained by the individual feeders, were withheld from all the animals on the anabolic steroid treatment until after the vial had been emptied. All rats received food and fresh water ad libitum.

One half of the animals in the exercised groups, normal and anabolic steroid treated, were sacrificed immediately after an exhaustive run. The other half of the trained animals and all of the control animals were sacrificed at rest.

At the time of sacrifice the animals were anaesthetized with ether (139) and their abdomens incised along the midline. The abdominal aorta was isolated and 10-12 mls of blood withdrawn into heparinized syringes. From this blood a 1.0 ml sample was taken for the determination of blood glucose while a 2.0 ml sample was used in determining blood lactate. Duplicate 1.0 ml samples were withdrawn from the remaining plasma for the determination of plasma FFA.

The epididymal fat pads were then removed, a 200-300

mg portion weighed and placed in a beaker containing 4.0 mls of Krebs-Ringer Solution and 0.20 gms of albumin for the determination of the release of adipose FFA. After the whole liver was removed and weighed a small weighed sample was placed at the bottom of a screw-cap test tube which was stored in a deep freeze (70°K) until it could be analyzed for glycogen content. The heart, biceps brachii and gastrocnemius muscles were then removed, weighed and stored in a deep freeze in a similar manner to await analysis for glycogen content. The spleen, adrenals, and testicles, when present, were also removed and weighed. All organ and tissue weights were measured on a Roller-Smith Precision Balance. The animals total body weight, taken immediately before sacrifice, was determined on a Triple Beam Balance.

Glycogen Determination

Liver and muscle glycogen was determined using the method developed by Lo, Russell, and Taylor (137). The frozen tissue samples of liver, heart, biceps brachii muscle and gastrocnemius muscle were first hydrolysed with 0.5 mls of 30% potassium hydroxide (KOH) containing saturated sodium sulphate (Na_2SO_4). The tissue samples, in capped test tubes, were then put into a boiling water bath for 20-30 minutes until a homogeneous solution was obtained after which they cooled in ice.

To precipitate out the glycogen from the alkaline digested solution 0.8 mls of 95% ethanol were added. After

standing in ice for half an hour or longer the samples were centrifuged at 840 x g for 20-30 minutes and the supernates carefully aspirated. The glycogen precipitate was then dissolved in 10.0 mls of distilled water. From this glycogen solution 1 ml was pipetted into a 150 x 20 mm test tube. One ml of 5% phenol solution was added to the above. To each test tube 5.0 mls of 96-98% sulphuric acid (H_2SO_4) were added rapidly, the stream of acid being directed against the liquid surface rather than against the side of the test tube for good mixing. After standing for 10 minutes the tubes were shaken and then placed in a water bath at 25°-30°C for 10-20 minutes before colorimetric readings were taken.

All samples were prepared in triplicate to minimize errors resulting from accidental contamination with cellulose lint. Absorbance readings for each of the triplicate samples were taken on a Beckman DU-2 spectrophotometer at a wavelength of 490 mu. The glycogen content of the samples was then subjected to the same phenol and sulphuric acid reactions as the muscle samples and the average readings of absorbance were plotted against the amount of glycogen used to produce a standard curve. During analysis a standard sample was run as a daily check on the standard curve. (Appendix A).

Blood Glucose Determination

Blood glucose was determined by the Nelson-Somogyi

method (39, 152, 179, 180). An alkaline copper solution was heated with protein-free filtrate whereby the blood sugar reduces cupric hydroxide to cuprous oxide. Arsenomolydate colour reagent was then added to reoxidize the copper. This was followed by the addition of distilled water to each tube of unknown sample. All test tubes were thoroughly mixed and allowed to stand for 15 minutes. Standard, blank and control specimens were prepared to assist in calculating the concentration of glucose. The optical density for each tube was read at a wavelength of 540 mu on a Beckman DU-2 spectrophotometer after zero optical density had been calibrated by means of the "blank". Glucose was then calculated in milligrams per 100 mls of blood.

Blood Lactate Determination

Blood lactate was determined by the Sigma Kit method (177). Immediately after the blood sample had been taken 2 mls of whole blood was transferred to a test tube containing 4 mls of cold 8% perchloric acid (PCA), a solution used in precipitating out proteins. The tubes were allowed to stand for 5 minutes and then were centrifuged at 3,000 rpm for 5 minutes to obtain a protein free filtrate. This filtrate was transferred to another test tube and recentrifuged to obtain a clear filtrate. A reagent containing lactic dehydrogenase suspension (LDH), glycine-hydrozine buffer, β-diphosphopyridine nucleotide (β-DPN) and distilled

water was mixed in a 125 ml erlenmeyer flask.

A series of test tubes were then labeled for analysis. The first six tubes were used to construct a standard curve. The remaining test tubes were set up in duplicate to consider values for unknown samples. To each of the tubes 2.9 mls of the reagent mixture was added. If the animal was sacrificed at rest 0.2 mls of the protein free filtrate were added to the test tube. If the animal was sacrificed after an exhaustive exercise bout 0.1 mls of the protein free filtrate was added to the test tube. These tubes were mixed well and allowed to stand for 45 minutes. The optical density for each tube was then read at a wavelength of 340 mu on a Beckman DU-2 spectrophotometer using the first test tube in the standard curve to calibrate for zero optical density. The values obtained for the unknowns were extrapolated from the standard curve to obtain blood lactate in milligrams per cent. (Appendix A).

Plasma and Adipose Free Fatty Acid Determination

Plasma FFA content was assayed by the method of Dole and Meinertz (47) while the release of FFA by the fat pads was estimated as outlined by Schotz and Page (173).

Because of the high correlation between plasma and adipose tissue FFA values observed by Gollnick (68) it was decided not to examine adipose tissue FFA homogenates.

In determining plasma FFA, duplicate 1 ml samples of

fresh plasma were added to 5 ml of fat extraction mixture in screw-cap tubes. The tubes were shaken and allowed to stand for 5 minutes. Two milliliters of distilled water and 3 ml of heptane were then added, the contents mixed thoroughly, and the tubes allowed to stand for 10 minutes. Three milliliter aquilots of the upper phase were taken and transferred to a 15 ml conical tube containing 1 ml of thymol blue working solution. Nitrogen was blown through a column containing sodium hydroxide (NaOH) to eliminate CO_2 and was bubbled through the sample while titrating as a means of agitation. A blank and standards were run for each set of determinations and a standard curve constructed. The plasma FFA concentration was calculated by taking the reading from the standard curve and multiplying by three. (Appendix A).

The release of FFA was estimated by incubating approximately 300 milligram samples of epididymal fat pads (173). After placing the weighed sample in a beaker containing 4.0 mg of Krebs-Ringer solution and 0.30 grams of albumin 1 ml aquilots were taken before and after 3 hours of incubation at 37°C in a Dubnoff metabolic shaker. The amount of FFA in the incubate was then determined by the method outlined by Dole and Meinertz (47). The release of FFA was then calculated in the same manner as plasma FFA.

Statistical Analysis

The data was analyzed using the one-way analysis of

variance. Newman-Keuls Multiple Comparison Between Ordered Means was used to test the difference between mean scores (211). Differences at the 0.05 level of confidence were considered to be significant.

CHAPTER IV

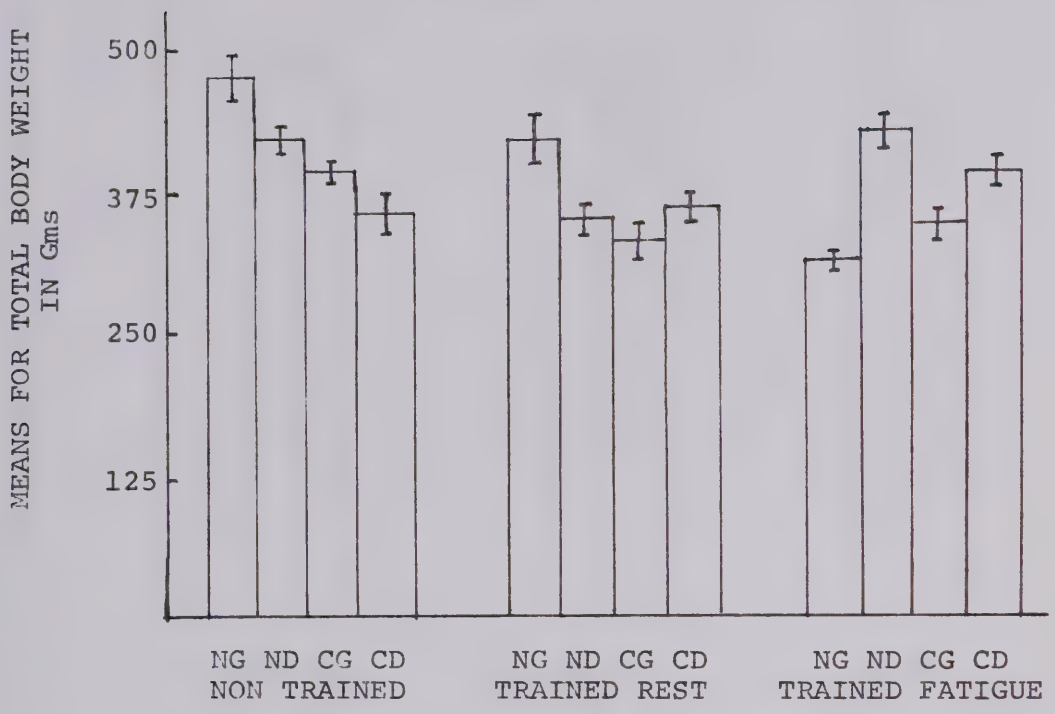
RESULTS

Neither training nor anabolic steroid treatment had a significant effect on total body weight (Figure 1)*. Significantly lower body weights, however, were observed when the normal trained animals were exercised to exhaustion. Castrated animals tended to have lower body weights than their noncastrated controls.

Organ weights of trained animals demonstrated that training had no significant effect on testicle, liver, spleen, or heart weights (Figures 2 to 6). Smaller liver weights, in those animals with anabolic steroid treatment, were noted for both the trained and nontrained groups. Anabolic steroid treatment, however, had no apparent affect on heart, spleen or testicle weights. Liver weights were lower in the fatigue groups and the castrated groups, while spleen weights were decreased in all groups after an exhaustive run. A slight increase of adrenal weights in the trained animals over the control animals was noticed (Figure 7). The adrenal weights of those animals exercised to exhaustion before sacrifice although heavier were not significantly greater.

*Tables for "Newman Keuls" Multiple Comparison Between Ordered Means are found in Appendix C.

FIGURE 1



LEGEND FOR ALL GRAPHS

NG: non castration, non anabolic steroid
ND: non castration, anabolic steroid
CG: castration, non anabolic steroid
CD: castration, anabolic steroid

TABLE 1

ANALYSIS OF VARIANCE FOR TOTAL BODY WEIGHTS

SOURCE	SS	DF	MS	F
TREATMENTS	0.122	11	11061.45	10.55*
EXPERIMENTAL ERROR	0.440	42	1048.74	
TOTAL	0.562	53		

*P<0.01

FIGURE 2

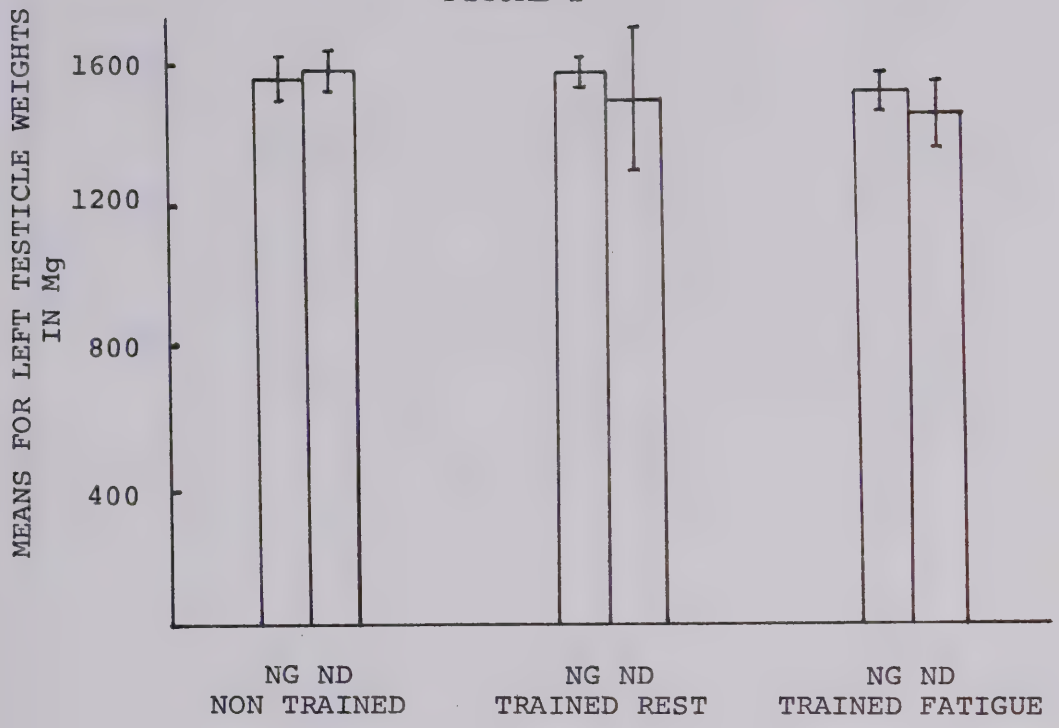


TABLE 2

ANALYSIS OF VARIANCE FOR LEFT TESTICLE WEIGHTS

SOURCE	SS	DF	MS	F
TREATMENTS	0.217	5	4342.40	0.06***
EXPERIMENTAL ERROR	0.142	21	67704.37	
TOTAL	0.359	26		

*** not significant at 0.05 level.

FIGURE 3

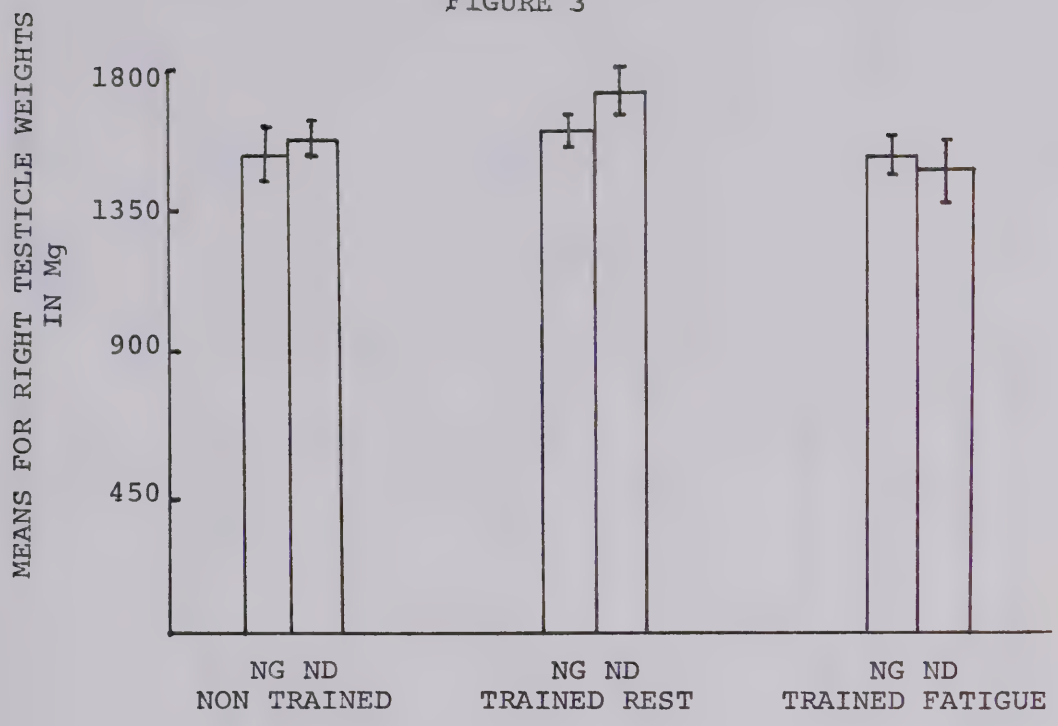


TABLE 3

ANALYSIS OF VARIANCE FOR RIGHT TESTICLE WEIGHTS

SOURCE	SS	DF	MS	F
TREATMENTS	0.147	5	29372.80	0.96***
EXPERIMENTAL ERROR	0.640	21	30495.23	
TOTAL	0.787	26		

*** not significant at 0.05 level.

FIGURE 4

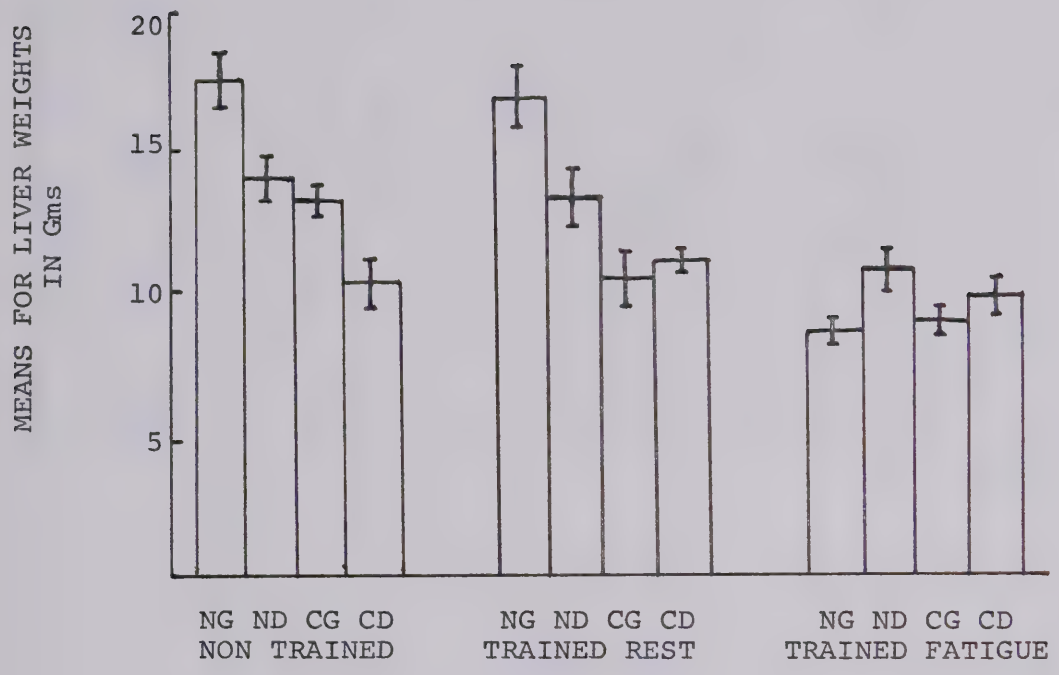


TABLE 4

ANALYSIS OF VARIANCE FOR LIVER WEIGHTS

SOURCE	SS	DF	MS	F
TREATMENTS	0.396	11	35.96	15.13*
EXPERIMENTAL ERROR	0.998	42	2.38	
TOTAL	1.394	53		

*P<0.01

FIGURE 5

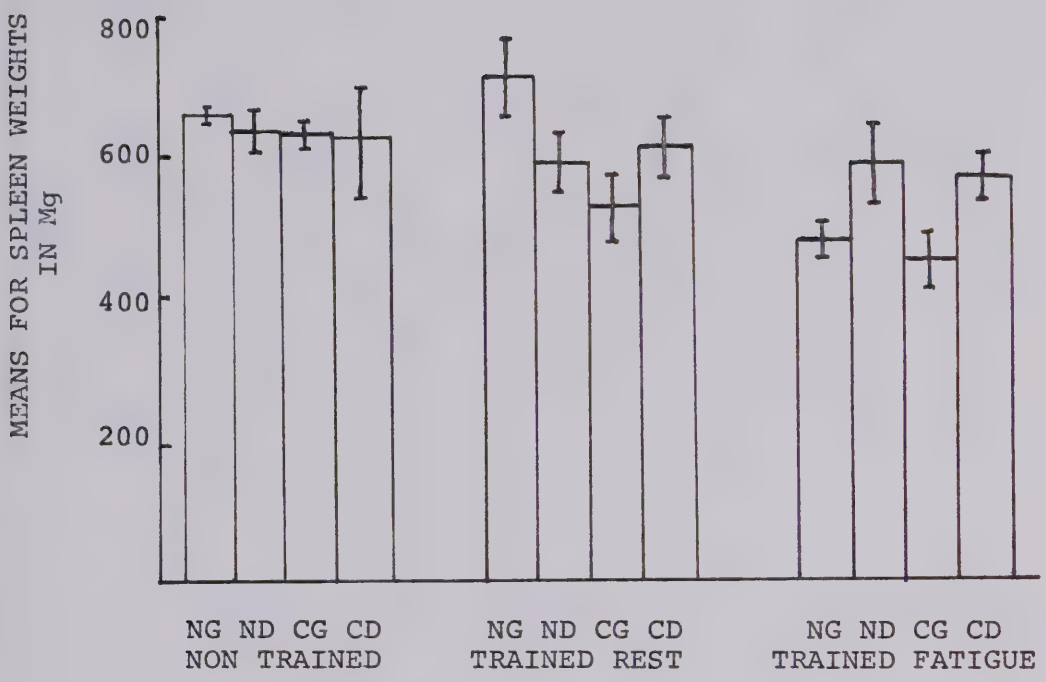


TABLE 5

ANALYSIS OF VARIANCE FOR SPLEEN WEIGHTS

SOURCE	SS	DF	MS	F
TREATMENTS	0.242	11	21988.36	2.60*
EXPERIMENTAL ERROR	0.355	42	8444.57	
TOTAL	0.597	53		

*P<0.01

FIGURE 6

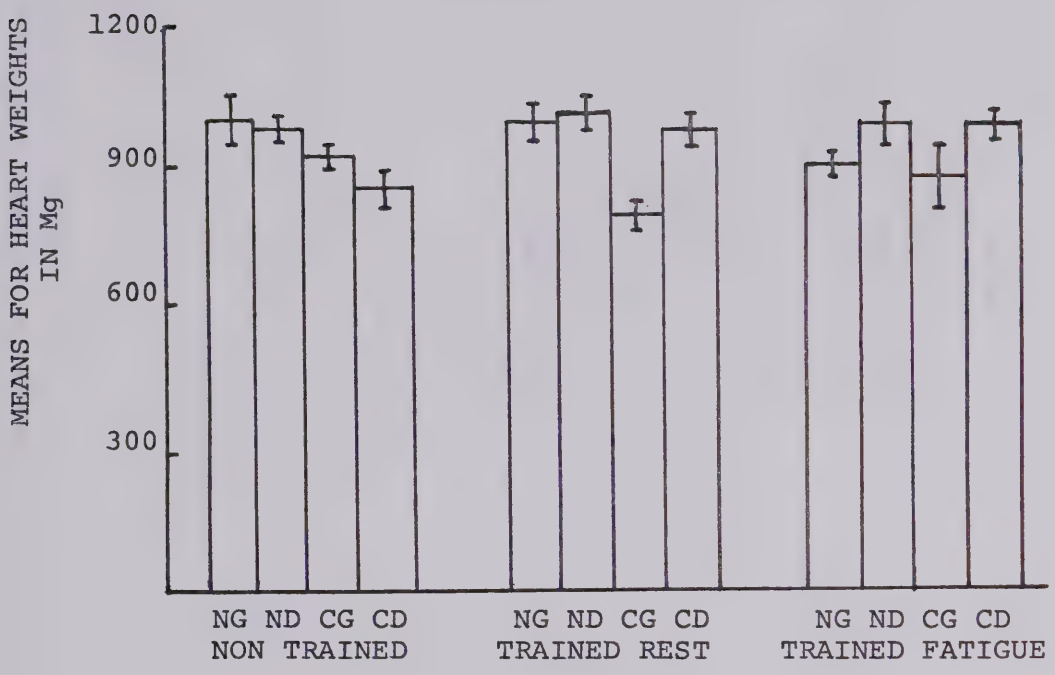


TABLE 6

ANALYSIS OF VARIANCE FOR HEART WEIGHTS

SOURCE	SS	DF	MS	F
TREATMENTS	0.430	11	39067.63	5.70*
EXPERIMENTAL ERROR	0.288	42	6851.05	
TOTAL	0.718	53		

*P<0.01

FIGURE 7

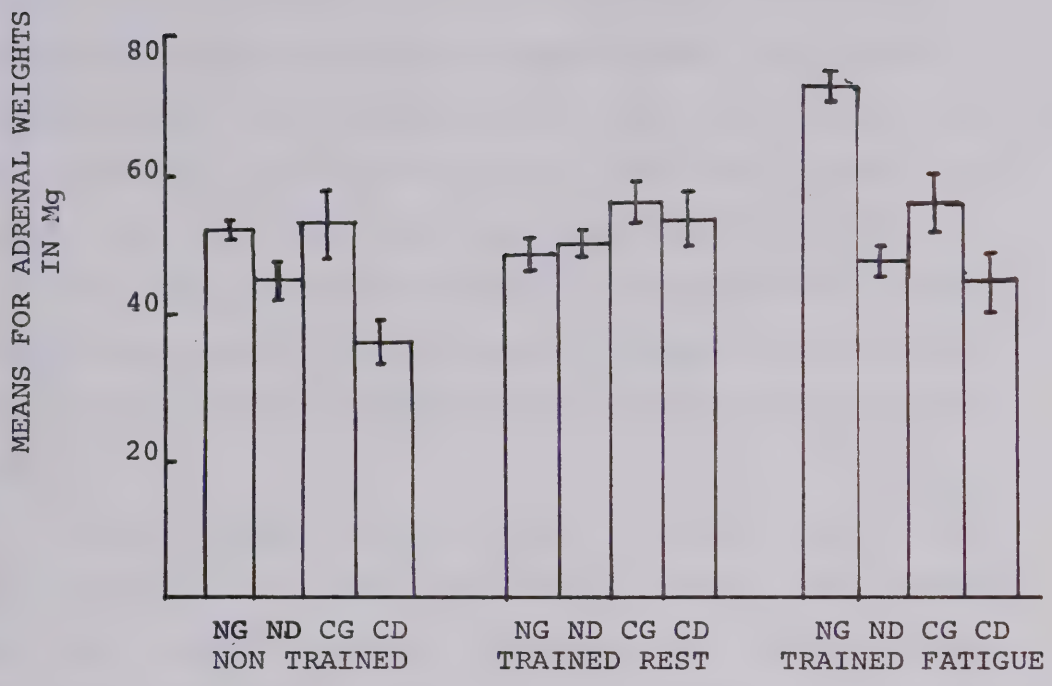


TABLE 7

ANALYSIS OF VARIANCE FOR ADRENAL WEIGHTS

SOURCE	SS	DF	MS	F
TREATMENTS	0.343	11	311.76	5.02*
EXPERIMENTAL ERROR	0.261	42	62.15	
TOTAL	0.604	53		

*P<0.01

Skeletal muscle glycogen levels of the normal and anabolic steroid treated animals indicated that in the groups sacrificed at rest, glycogen content was similar for the trained and sedentary rats (Figures 8 and 9). In the nontrained and trained animals sacrificed at rest, glycogen stores decreased with anabolic steroid treatment. Castration had no apparent effect on carbohydrate stores. Exhaustive exercise significantly increased the depletion of glycogen from the biceps brachii and gastrocnemius muscles.

Liver glycogen stores were not affected by training nor castration but were significantly reduced after exhaustive exercise (Figure 10). Cardiac muscle glycogen stores were not affected by either training, anabolic steroid treatment, fatigue or castration (Figure 11).

Blood glucose concentrations revealed that no differences existed between trained and nontrained groups (Figure 12). Although blood glucose levels decreased with exercise in most cases only those animals in the castrated, anabolic steroid treated groups had significantly lower values.

No differences existed for resting blood lactates in the normal and anabolic steroid treated animals (Figure 13). Exercise caused the blood lactate levels to rise with a sharp increase occurring in the castrated groups. Anabolic steroid treatment, however, had no apparent effect on blood

FIGURE 8

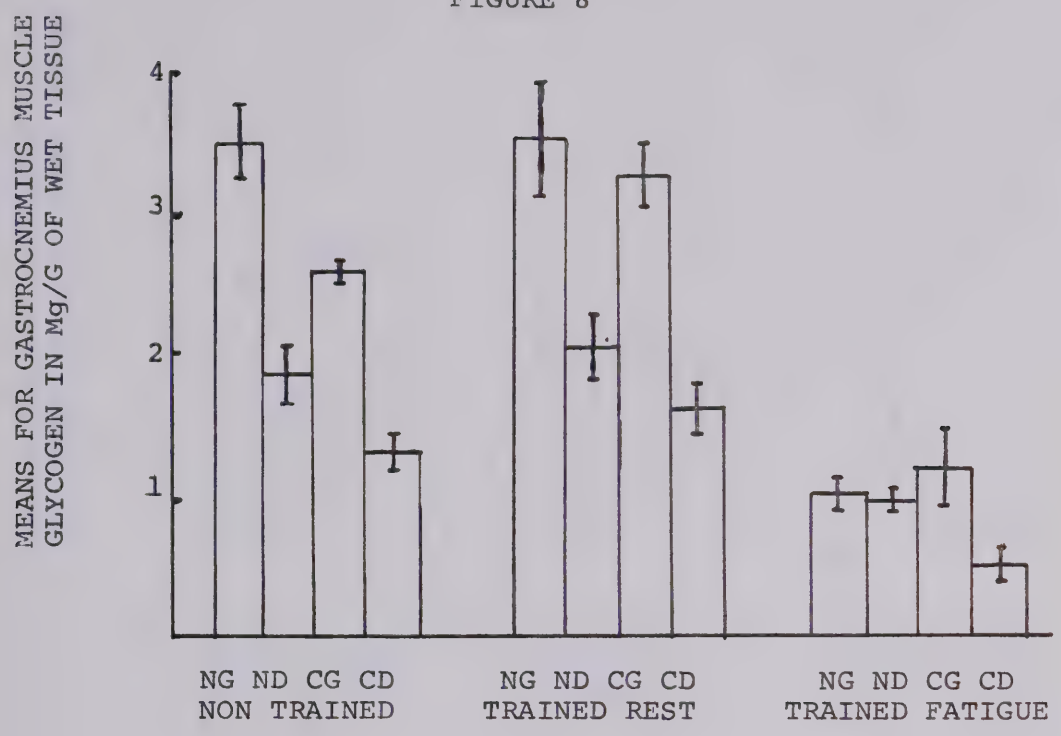


TABLE 8

ANALYSIS OF VARIANCE FOR GASTROCNEMIUS MUSCLE GLYCOGEN

SOURCE	SS	DF	MS	F
TREATMENTS	0.489	11	4.44	17.85*
EXPERIMENTAL ERROR	0.106	42	0.25	
TOTAL	0.595	53		

*P<0.01

FIGURE 9

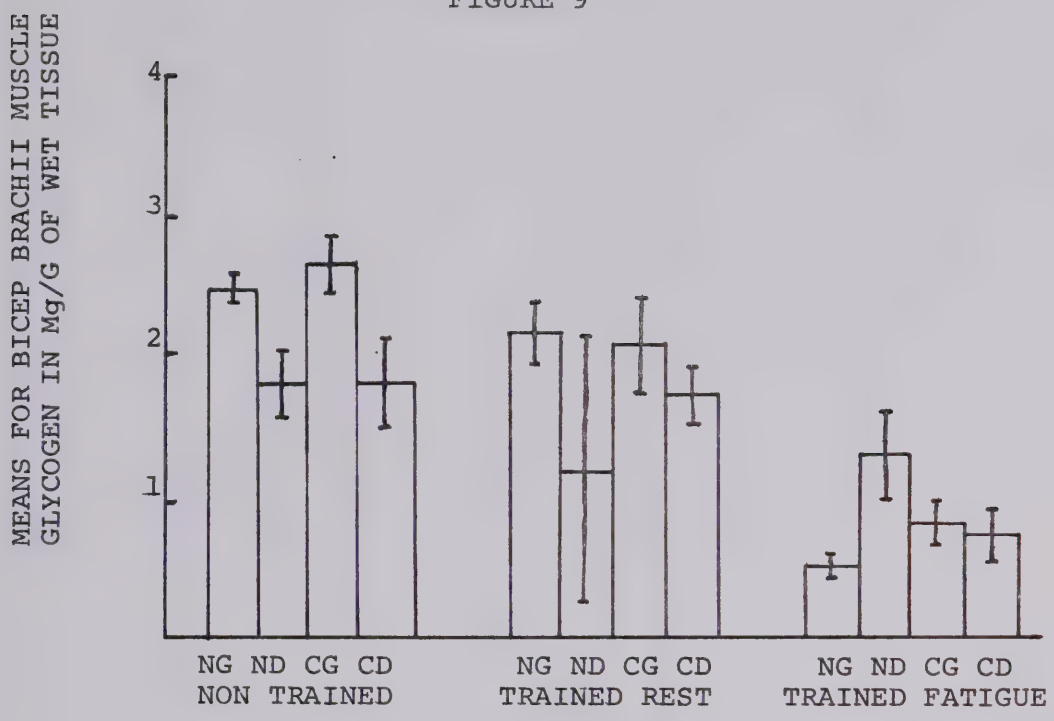


TABLE 9

ANALYSIS OF VARIANCE FOR BICEP BRACHII MUSCLE GLYCOGEN

SOURCE	SS	DF	MS	F
TREATMENTS	0.225	11	2.05	8.66*
EXPERIMENTAL ERROR	0.996	42	0.24	
TOTAL	1.221	53		

*P<0.01

FIGURE 10

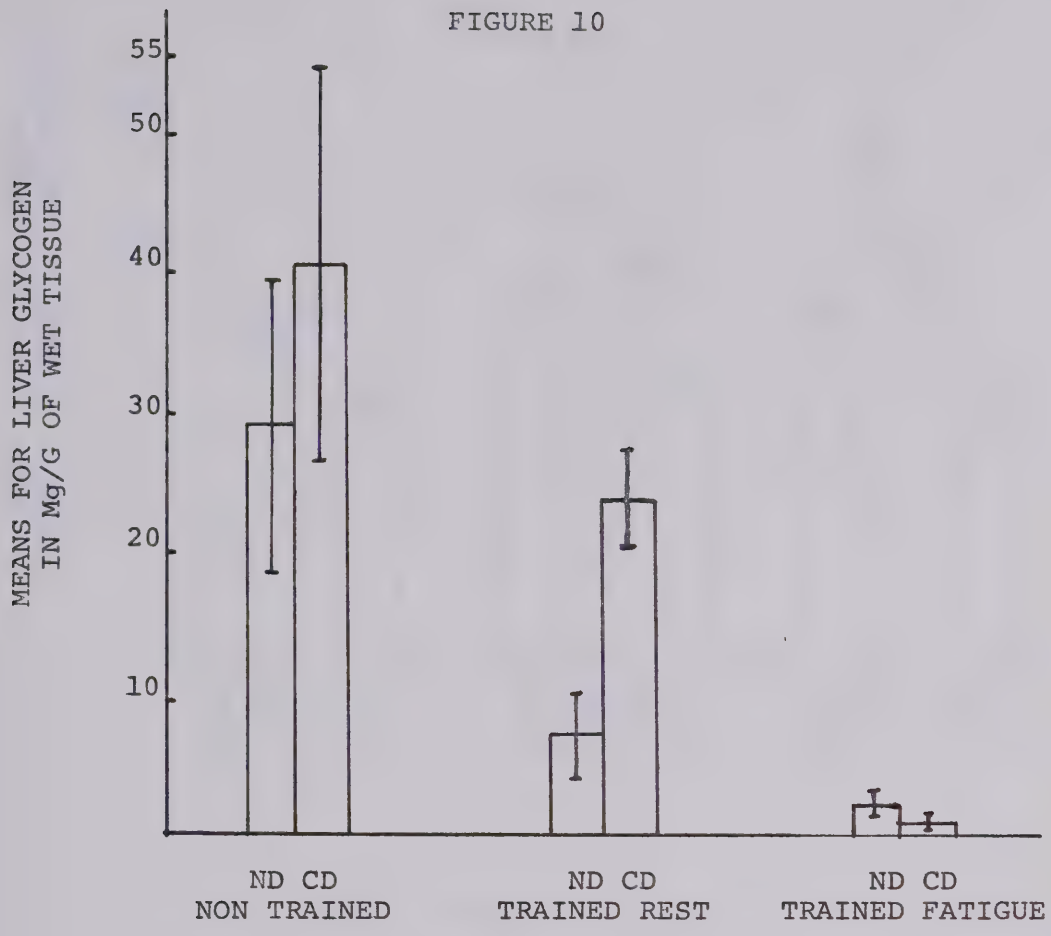


TABLE 10

ANALYSIS OF VARIANCE FOR LIVER GLYCOGEN

SOURCE	SS	DF	MS	F
TREATMENTS	0.574	5	1348.66	3.45**
EXPERIMENTAL ERROR	0.938	24	390.70	
TOTAL	1.612	29		

**P<0.05

FIGURE 11

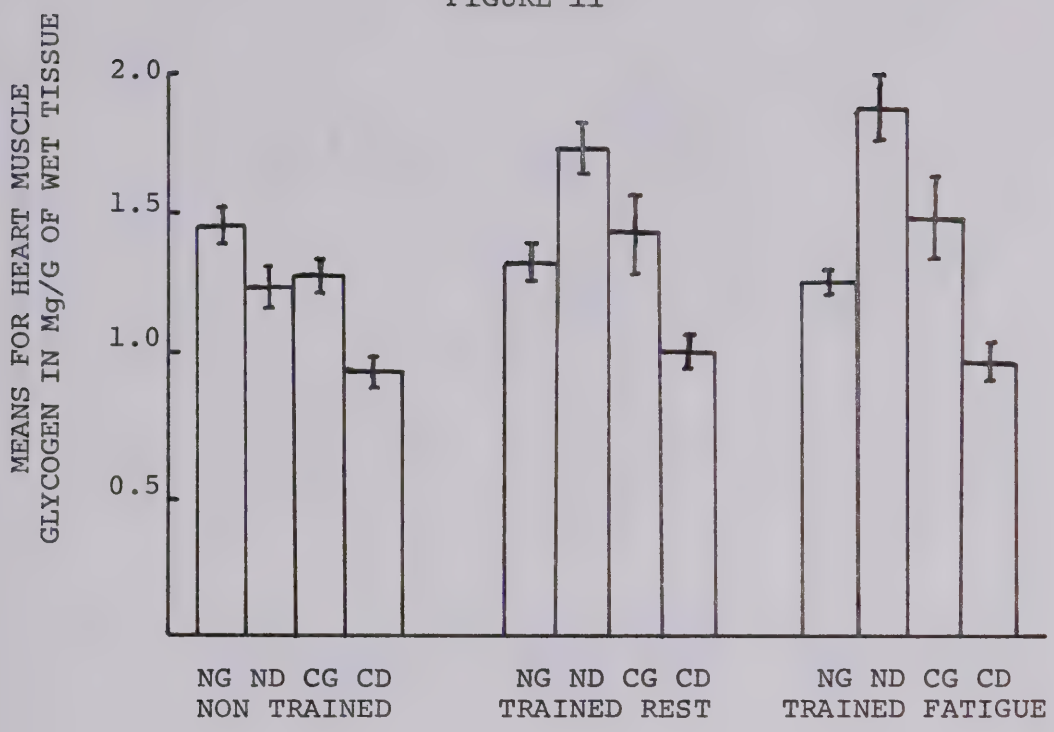


TABLE 11

ANALYSIS OF VARIANCE FOR HEART MUSCLE GLYCOGEN

SOURCE	SS	DF	MS	F
TREATMENTS	0.415	11	0.38	2.36**
EXPERIMENTAL ERROR	0.672	42	0.16	
TOTAL	1.087	53		

**P<0.05

FIGURE 12

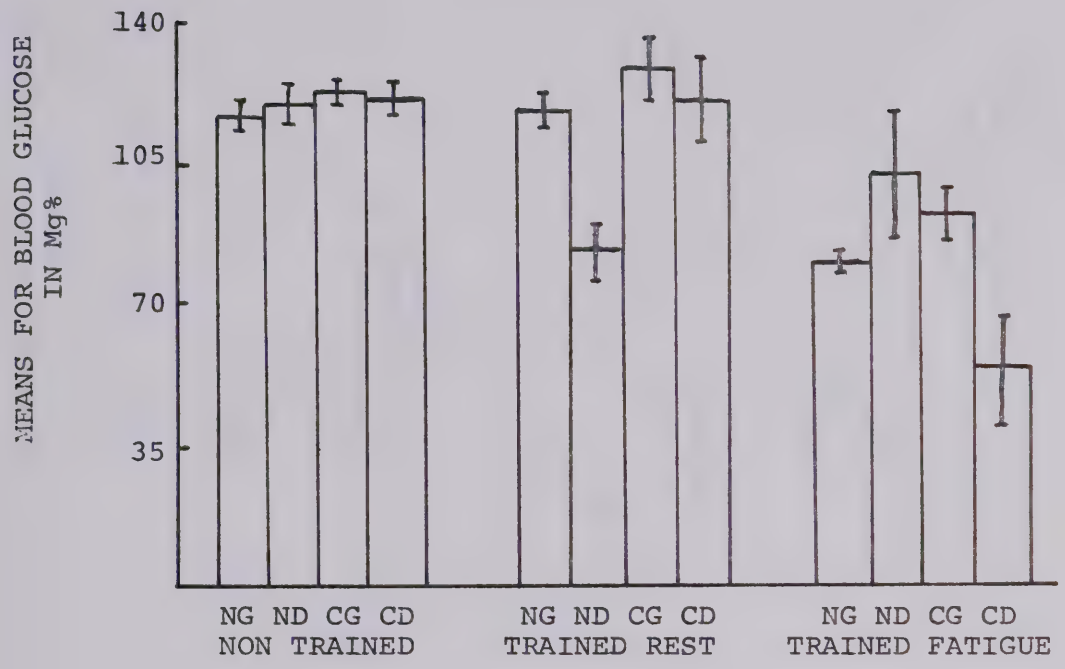


TABLE 12

ANALYSIS OF VARIANCE FOR BLOOD GLUCOSE

SOURCE	SS	DF	MS	F
TREATMENTS	0.268	11	2436.03	6.26*
EXPERIMENTAL ERROR	0.163	42	388.85	
TOTAL	0.431	53		

*P<0.01

FIGURE 13

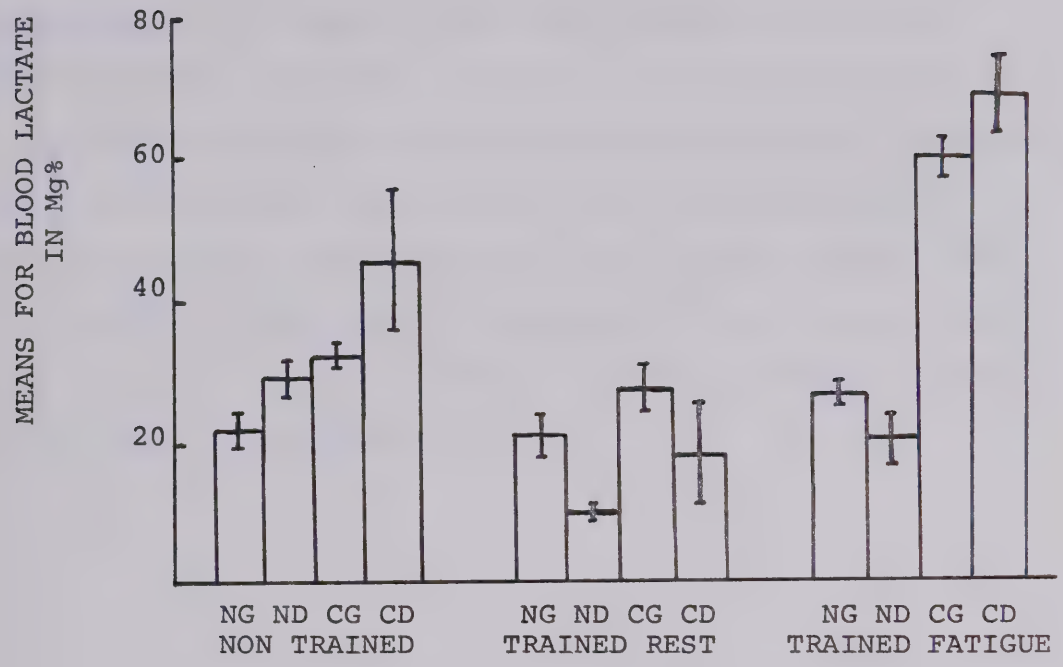


TABLE 13

ANALYSIS OF VARIANCE FOR BLOOD LACTATE

SOURCE	SS	DF	MS	F
TREATMENTS	0.157	11	1423.40	11.21*
EXPERIMENTAL ERROR	0.533	42	127.02	
TOTAL	0.690	53		

*P<0.01

lactate levels.

Plasma and adipose tissue FFA analysis indicated no difference in the resting FFA levels between control and trained animals (Figures 14 and 15). Exercise produced a significant increase in plasma FFA concentration. Although mobilization of FFA from adipose tissue occurred during exhaustive exercise in normal rats, the anabolic steroid treated animals were not able to mobilize FFA as readily. Castration did not appear to have a significant effect on the concentration of adipose tissue FFA.

FIGURE 14

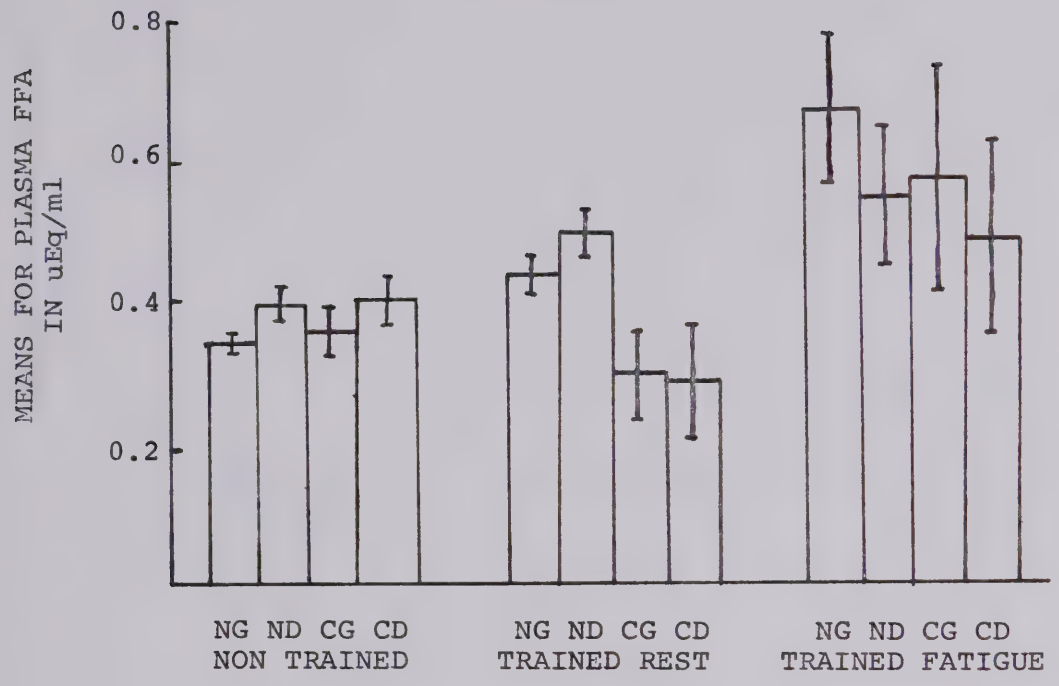


TABLE 14

ANALYSIS OF VARIANCE FOR PLASMA FFA

SOURCE	SS	DF	MS	F
TREATMENTS	0.666	11	0.06	7.17*
EXPERIMENTAL ERROR	0.355	42	0.01	
TOTAL	1.021	53		

*P<0.01

FIGURE 15

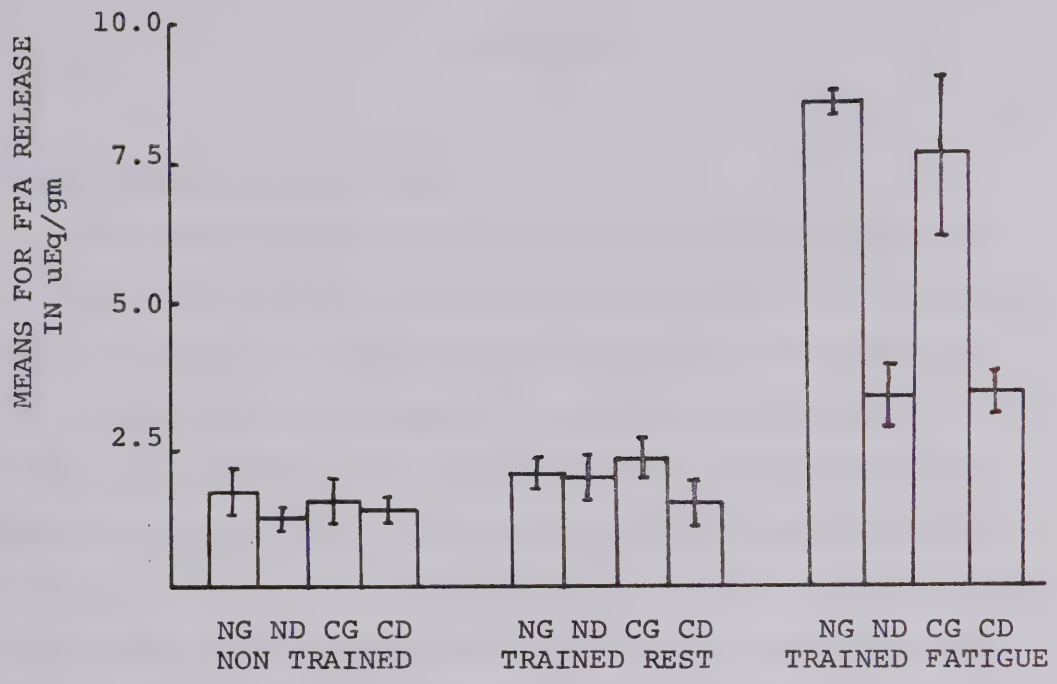


TABLE 15

ANALYSIS OF VARIANCE FOR FFA RELEASE

SOURCE	SS	DF	MS	F
TREATMENTS	0.284	11	25.84	26.86*
EXPERIMENTAL ERROR	0.404	42	0.96	
TOTAL	0.688	53		

*$P < 0.01$

CHAPTER V

DISCUSSION

Total Body and Organ Weights

Evidence relating to fluctuations in the body weight of laboratory animals following regular exercise is conflicting. Several investigators (52, 182, 185) have reported that regular exercise causes a decreased body weight in laboratory animals when compared with nontrained controls. Steinhaus et al (182) and Hearn et al (88) attributed this finding to differences in the amount of fat in the two groups. These results were noted when the animals were exercised both by running and by swimming. Other researchers have reported either increases in body weight following regular exercise (82, 158) or no significant changes (88, 144, 193).

The results of this study which showed less of an increase in body weight for trained animals, for the most part, concur with the findings of Hearn et al (88), McClintock et al (144), and Tepperman et al (193). Stevenson et al (185) found that the weight difference was approximately proportional to the amount of exercise performed by the animals. Therefore, the differences in results obtained in the present investigation and those of Eranko et al (52), Steinhaus et al (182), and Stevenson et al (185) may be due to variations in training programs. The decrease in total

body weight following exhaustive exercise might be attributed to the decrease in glycogen levels, the mobilization of fats and the release of stored blood cells.

Kochakian et al (120-126) reported decreases in total body weights of laboratory animals following castration. This loss was attributed primarily to atrophy of skeletal muscles. Since muscle size was not examined in the present study the contention of Kochakian et al could not be confirmed.

Androgens, the male sex hormones producing masculine characteristics, are the primary hormones of the testes. Androgens have protein anabolic effects and readily induce a positive nitrogen balance. Testosterone is the chief testicular androgen and is secreted by the Leydig cells (149). The increase in weight of the adolescent is due primarily to the marked increase in muscle mass as induced by the increase in circulating androgens (154). If the testes are removed there is permanent sterility. Owing to absence of testosterone the usual pubertal changes do not occur and the accessory organs of reproduction do not develop. In addition, the muscles are soft and poorly developed (116). Since prepubertal castration was performed on the animals in this investigation, thereby removing the major source of androgens, it is possible that the loss in body weight may have been due to atrophy or nondevelopment of skeletal muscle.

Proper treatment with androgen reverses many of the physical changes induced by prepubertal castration (154). Kochakian et al (120, 124) observed that involution of skeletal muscles after castration can be reversed by the administration of androgens. A gain in weight beyond the normal, however, was only effected by copious dosages of androgens (121). Since the loss in body weight caused by castration was not affected by anabolic steroid treatment it would appear that the dosage of methandrostenolone employed in this study was not sufficient to alter the changes produced by castration.

After the administration of anabolic steroids testicular weight has been found to decrease due to atrophy (51, 150). Results of the present study, however, indicated that testicular weight was not affected by anabolic steroid treatment. This apparent discrepancy from the literature might be explained by the relatively lower dosage of anabolic steroid used in this investigation.

Research on the effect of training on liver weight is conflicting. Asahino et al (8) and Donaldson and Meeser (49) have reported decreases while Hatai (82) reported increases in rats following regular exercise. After an increase of liver weight in the first 20 days of exercise Asahino et al (8) noted that further training produced liver atrophy accompanied by cell infiltration and hyperplasia of interlobular connective tissue. Upon an examination

of regressed liver weights Bloor et al (22) reported that cellular composition of the liver was different in the trained compared to the controls. Cytoplasmic mass of individual hepatic cells was increased in the trained groups. This finding might account for the increase in liver weight with training found by Hatai (82).

The present investigation, in concurrence with the studies of Dahlgren (40) and Steinhaus et al (182) found no significant changes in liver weights after training. These different results might be attributed to the variation in intensity of training employed in each study. For example, the most strenuous exercise programs were used primarily in cases where a loss in liver weight was indicated (8, 49).

When animals were sacrificed after an exhaustive exercise bout liver weights were lower than those of the nonexercised controls (181). Similar results occurred in the present study. These findings have been attributed partially to a depletion of glycogen stores, with some mobilization of fat and the release of stored red blood cells contributing to the loss (181). Within the realm of this investigation it is possible that glycogen depletion is a major factor in liver weight loss.

A decrease in liver weight following castration has been reported by Talaat et al (187). This has been primarily attributed to a reduction in liver glycogen with a concomitent increase in the fat and water content of

the liver. The decrease reflected in this study may be partially attributed to a liver glycogen decrease. However, since liver fat and water content were not examined the assertion by Talaat et al (187) regarding these parameters cannot be substantiated.

Talaat et al (187) found that after repeated injections of testosterone proprionate increases in liver weight occurred accompanied by an increase in liver glycogen and a decrease in liver fat. Although anabolic steroid treatment appeared to reduce the size of the liver in the present study, this phenomenon remained unexplained.

It is generally accepted that exercise produces a decrease in spleen weight (16, 74, 182, 194). Many factors have been postulated to explain this decrease, however, existing evidence only supports the fact that this is a result of the mere expulsion of storage blood (16, 73). Results of the present investigation demonstrate that exercise to fatigue significantly reduced the weight of the spleen. This finding confirms the data reported by Barcroft et al (16), Gollnick et al (74), Steinhaus et al (182) and Tipton et al (194).

Kimeldorf and Baum (118) and Bloor et al (22) have associated a heavier spleen with trained animals. This size difference of the spleen was due to an increased mass of lymphoid tissue (22). Although Gollnick (67) and Montoye et al (147) have reported decreases in absolute spleen

weights following training, Hearn (87) and Tipton et al (194) demonstrated no differences in spleen weight between nontraine, trained or detrained rats. Training did not significantly affect the absolute weight of the spleen in the present study.

Regular endurance exercise, including swimming has previously been shown to produce cardiac hypertrophy in rats and other animal species (22, 79, 193, 197, 198) as well as in humans (14). In 1915 Hatai (82) reported cardiac enlargement in rats that had voluntarily exercised in a revolving cage. The variability in the activities of the animals in the revolving cage suggested that there might exist a definite relationship between heart weight and the amount of exercise performed. Dahlgren (40) suggested that the intensity of her training program was not of sufficient magnitude to elicit a change in heart weight of trained rats. Results of the present study indicate that training had little effect on the animals' heart weight.

Although evidence by Brown et al (24), Eagan et al (51) and Murphy et al (150) showed an increase in heart weight following anabolic steroid treatment results from the present study, which found no changes, does not substantiate their findings. This discrepancy might be due either to the lower dosage of anabolic steroid used in this study or to the training programs, or a combination of

these factors.

There is general concensus in the literature that training produces increased adrenal weights (67, 70, 74, 88). This study, in concurrence with Bloor's investigation (22), revealed only slight increases of adrenal weights in the trained animals over the control animals. Bloor et al (22) found the histological examination of the adrenals in the exercised groups showed an increased amount of cytoplasm per adrenal cortical cell, suggesting an increased glucocorticoid secretion in the exercised animals.

Muscle and Liver Glycogen

Increases in skeletal muscle glycogen content following regular exercise programs have been reported by many investigators (21, 72, 76, 133, 159, 190). This elevation in glycogen content may be important in increasing work capacity during heavy exercise (20, 76). Procter and Best (159) contend that there may be an optimum period of training for the accumulation of glycogen since a program of more than four weeks produces no significant changes in glycogen stores. Similar increases of glycogen levels have been reported in cardiac tissue after training for two 1 hour periods at 1 mile per hour each day (50). The results of this study demonstrated that training had little effect on glycogen levels. This finding does not agree with earlier reports by Gollnick et al (72), Grollman (76),

Procter and Best (159) and Taylor et al (190).

Evidence confirms that a depletion of glycogen stores occurs during prolonged exercise (72, 133, 181, 190, 205). This loss of glycogen from the liver and skeletal muscle during an exhaustive run has been interpreted as meaning that the glycogen is being utilized to provide energy for the performance of work (2, 205). The present investigation found results similar to those reported in the literature. Values noted in this study, however, were slightly higher than those reported in the literature (72, 181).

Castration has been known to be followed not only by weight loss but also by an initial decrease in glycogen levels of rat skeletal muscles (19, 187). These levels, however, returned to normal after approximately forty days (19, 187). Castration also had similar effects on liver glycogen levels (187). In this study castration had no apparent effect on carbohydrate stores after 90 days.

The administration of testosterone and its derivatives increase glycogen levels in both normal and castrated animals quite apart from pituitary influence (135, 187). Both Taylor et al (191) and Talaat et al (187) reported no changes in muscle glycogen after prolonged use of testosterone proprionate. Liver glycogen stores, however, were increased significantly (187). The present study found that muscle glycogen stores were lower in the anabolic steroid treated animals than in the normal animals.

Blood Glucose

Exercise effects on plasma glucose are known to be directly influenced by the duration, intensity and type of work. Exercise of light to moderate intensity, short duration and therefore of relatively aerobic nature results in little change in plasma glucose concentration (91, 167). At the onset of activity an increase in lactate concentration and carbon dioxide production is noted but the increase relative to blood glucose changes shows the origin is probably from muscle glycogen (165).

Although plasma glucose concentration is relatively unchanged a turnover in glucose is constantly occurring. Despite the vast changes that can occur in the hepatic output of glucose resulting from exercise investigators have hypothesized that it is the uptake of glucose by the muscle cell that is the primary factor in determining plasma glucose levels (102, 165, 172).

Since FFA mobilization and utilization is inhibited by increasing lactate concentration during short duration exhaustive work glucose becomes a very important source of energy to the muscle (56, 203). Wahren et al (204) found that in heavy exercise, blood glucose may account for 75-89% of the estimated total carbohydrate metabolism and concluded that glucose becomes more important as exercise reaches maximal levels. In conjunction with prolonged activity blood glucose levels have been observed

to fall both in man and in animals (26, 72, 136, 164, 168, 212). Issekutz and Allen (106) studied normal dogs during prolonged treadmill exercise and noted a progressive decline in blood glucose levels. Similar trends were recorded in the present study.

The general trend of blood glucose response to exercise is proposed to follow the pattern of a decrease immediately at the onset of exercise, a subsequent gradual increase as exercise is continued and a decrease as exercise is prolonged further. In each of these activity states the blood glucose concentration is finely regulated within the limits demanded by the individuals needs. This regulation is via specific humoral-chemical factors.

Talaat et al (187) have reported a decrease in fasting glucose levels up to 25 days after castration. There was also a reduction in glucose tolerance, in sensitivity to insulin and the urinary output of 17-ketosteroids. Since liver and muscle glycogen stores are major sources of glucose a decrease in their levels may account for lower blood glucose levels following castration. These variables returned to normal 30 days following castration. This might explain the results obtained in this study which found no significant differences in glucose levels 90 days after castration.

Talaat et al (186, 187) also noted that the effect of single or repeated injections of testosterone proprionate

in castrated animals depended on the time since castration. That is, injections of either 1 or 5 mg/kg of testosterone proprionate administered for 7 days in animals castrated 7 days previously could counteract the effect of castration on blood glucose. On the other hand, when the male hormone was administered for 30 days or more starting immediately after castration the effect of orchiectomy remained evident and blood glucose was still low. This could account for the low blood glucose levels in the anabolic steroid treated castrated rats in the present investigation. It therefore appears that time since castration is an important factor to take into consideration when studying the effects of steroids on blood glucose levels in castrated rats.

Blood Lactate

Lactate has long been known to be a product of exercise, and its existence in sufficient quantities in the blood plasma or muscle tissue is generally indicative of anaerobic metabolism. The formation of lactate from pyruvate provides storage for excess ions of carbohydrate metabolism and permits work to be carried out in the absence of oxygen. The accumulation of lactate is a limiting factor to exercise dependent on the level incurred in relation to the severity and duration of the activity (12).

The effects of training on blood lactate levels have

been extensively examined. Margaria et al (141), Barnard (17), Williams (210), and Cunningham et al (38) have reported that training causes a decrease in blood lactate responses for a given work load. This decrease has been attributed to the greater aerobic capacities developed by training and is best demonstrated in the changes of the lactate versus MVO_2 curve in trained and untrained individuals (12). Although training has appeared to decrease lactate levels in trained animals when compared to the controls in the present study the differences were not significant.

It has been reported by several investigators that in light or moderate exercise up to 40-50% of an individuals maximum aerobic capacity the oxygen supply to the tissue is sufficient for near-complete oxidation of pyruvic acid (12, 65, 89, 114, 151). Prolonged exercise of moderate intensity results in a preliminary rise in blood lactate followed by a later decline (11, 12, 37, 89). Astrand et al (11) and Costill (37) have reported a decline in lactate concentration over extended physical activity such that blood lactate was less than 70 mg% after 1-2 hours of exercise even when the exercise was of an intensity associated with increased lactate production.

Data from the present study, with the exception of castrated animals, indicates that the prolonged exhaustive activity was of such an intensity as to allow near-complete

pyruvate oxidation. Animals in the castrated groups, however, had significantly higher blood lactate levels. This might be due to the decreases in erythrocyte count and hemoglobin content of the blood resulting from castration (149, 195). Since hemoglobin allows for the carriage of large amounts of oxygen any decrease in its content would tend to lower the amount of oxygen available for metabolic activities.

Literature reviewed for this study indicated that an examination of the effects of anabolic steroids on blood lactate levels has not been carried out. Results of the present investigation show that blood lactate levels were not affected by anabolic steroids.

Free Fatty Acids

The importance of fat as an energy source, especially during aerobic muscular work, has been well documented (12, 46, 105). A review of the literature reveals that the mechanisms of control involved in the release and uptake of FFA are relatively complex and intricately interrelated. Fat is stored abundantly in the form of triglycerides in adipose tissue, primarily in the splanchnic regions, to a lesser extent around the muscle and finally intramuscularly (157). It has been shown that albumin-bound FFA can be readily oxidized by muscle, in vitro as well as in vivo (108), and is a major source of fuel for muscular work (61,

62, 157).

Gollnick et al (72) reported plasma FFA values of approximately 0.30 uEq/ml for groups of normal sedentary and trained rats sacrificed at rest. Similar results were noted in the present study. Froberg (63) found values of 0.38-0.43 uEq/ml for an identical sample. In humans, Taylor (189) has suggested resting levels of 0.50 uEq/ml $\pm$ 0.15 uEq/ml.

In experiments with dogs, Issekutz et al (108) demonstrated that trained animals have greater ability to utilize FFA as an energy source. Similarly, Miller et al (146) found increased FFA mobilization and decreased lactate concentration in trained dogs as compared to untrained animals. Paul (157) reported that, in trained dogs, aerobic exercise with little or no lactate accumulation shows a shifting of the metabolism to fat regardless of the energy expenditure. He attributed this to the better vascularization of the splanchnic region and other fat depot regions.

Adrenergic neuro-humeral activity, a factor playing an important role in the internal adjustment of the intact organism to exercise, has been shown to increase during exercise (53, 85). This has been found to affect the rates of lipolysis and release of FFA from the lipid store (12, 61, 62, 105). Evidence has only recently been reported indicating that nervous stimulation plays a greater role in FFA mobilization than catecholamine levels. Gollnick et al

(72) noted that in adrenalectomized and adrenomedullectomized rats that lipolysis was not inhibited during exercise, indicating that a release of catecholamine from the adrenal medulla is not essential for controlling lipolysis during exercise. After hypophysectomy, which eliminates noradrenergic hormones such as ACTH, growth hormone, TSH, adrenal glucocorticoids and glucagon, and beta-blockage a complete blockage of lipolysis occurred during exercise. They concluded, therefore, that both the adrenergic and noradrenergic systems exert an effect on lipid mobilization during exercise.

Rosell et al (169) compared the mobilization of FFA during electrical stimulation of the sympathetic nerves entering the fat pads and during intra-arterial infusion of catecholamines. After finding that FFA mobilization occurred by a nerve stimulation equivalent to that occurring at rest, while an equivalent mobilization occurred only by infusing an amount of catecholamine equivalent to that existing during severe stress it was concluded that nervous stimulation plays a greater role in FFA mobilization than catecholamine levels. Havel and Goldfien (85) have reported similar trends.

The presence of lactate, glucose, and insulin have been shown to cause inhibition of FFA mobilization (18, 30, 109, 168). Issekutz et al (108) reported that lactate infusion caused a significant decrease in the release,

uptake and rate of oxidization of FFA. Gold et al (66) found a similar effect.

Prolonged exercise has been shown to influence the uptake and utilization of FFA as well as their release from adipose tissues (59, 78, 113). Increased utilization of FFA during this type of exercise implies that mobilization and release of FFA by adipose tissue is also elevated by prolonged activity. The elevated plasma FFA levels and the release of FFA following prolonged exercise in the present study indicates that lipid mobilization in the fat depots is stimulated by muscular work. During prolonged activity FFA is released from the fat pads into the circulation systems of humans (57, 58), dogs (105), and rats (68). Light prolonged exercise increases plasma FFA (29, 30, 59, 102). During this type of exercise the blood flow to the adipose tissue is probably sufficient to transport the FFA as it is formed and released, thereby producing a gradual increase in plasma FFA content.

Taylor et al (191) have reported that castration had no significant effect on plasma FFA levels and FFA release. Although castration did not have a significant effect on FFA levels in the present investigation a slight decreasing trend was apparent. This might be caused by an elevation in lactate levels; a factor contributing to the inhibition of FFA mobilization (66, 107).

A review of the literature revealed that no material

was available concerning the relationship between FFA and anabolic steroid treatment. Results from the present study found that those animals treated with anabolic steroids did not elicit the increase in tissue FFA concentration noted in the normal rats. Although it would appear that anabolic steroids affect the release of FFA the mechanisms involved are not fully understood.

CHAPTER VI

SUMMARY AND CONCLUSION

Fifty-four albino rats (Wistar strain) were used to determine the effect of prolonged use of anabolic steroids on energy sources of trained and nontrained rats.

Animals were divided into two equal groups: one group was bilaterally castrated while the second served as non-castrated controls. One half of each group received Dianabol (Methandrostenolone - 17α - methyl - 17β - hydroxy-androsta - 1, 4 - dien - 3 - one) orally administered weekly (1.75 mg/kg). Two thirds of each group were trained to exercise on a motor driven rodent treadmill by progressively increasing the speed and duration of the daily exercise session until they were capable of running continuously for one hour at a speed of 1 mph. Each animal then continued to run at this intensity for an additional 8-9 weeks. One half of the trained animals were sacrificed immediately after a fatiguing run while all other animals were sacrificed at rest.

Analysis of the results indicated that regular exercise had no effect on the parameters measured with the exception of adrenal weights which increased slightly as a result of training. When the animals were exercised to exhaustion decreases occurred in liver and spleen weights,

skeletal muscle and liver glycogen, and blood glucose levels. Increases were apparent in adrenal weight, blood lactate, plasma FFA and lipid FFA mobilization. Analysis also revealed that castration caused decreases in body and liver weights as well as increases in blood lactate levels.

Anabolic steroid treatment did not affect body weight, testicular size, spleen, heart, adrenal weights, blood lactate, resting blood glucose levels and resting FFA levels. Decreases, however, were noted in liver weights and resting skeletal muscle glycogen levels. When the animals were exercised to exhaustion it was apparent that anabolic steroids had no effect on the depletion of glycogen stores or on the utilization of blood glucose. Although mobilization of FFA from adipose tissue occurred during exhaustive exercise in normal rats, the anabolic steroid treated animals were not able to mobilize FFA as readily.

Conclusion

Within the limitations of this study it can be stated that anabolic steroids had a significant effect on skeletal muscle glycogen sources and the mobilization of FFA sources in exercised trained animals.

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APPENDIX A

STANDARD CURVES

FIGURE 16

STANDARD CURVE FOR GLYCOGEN

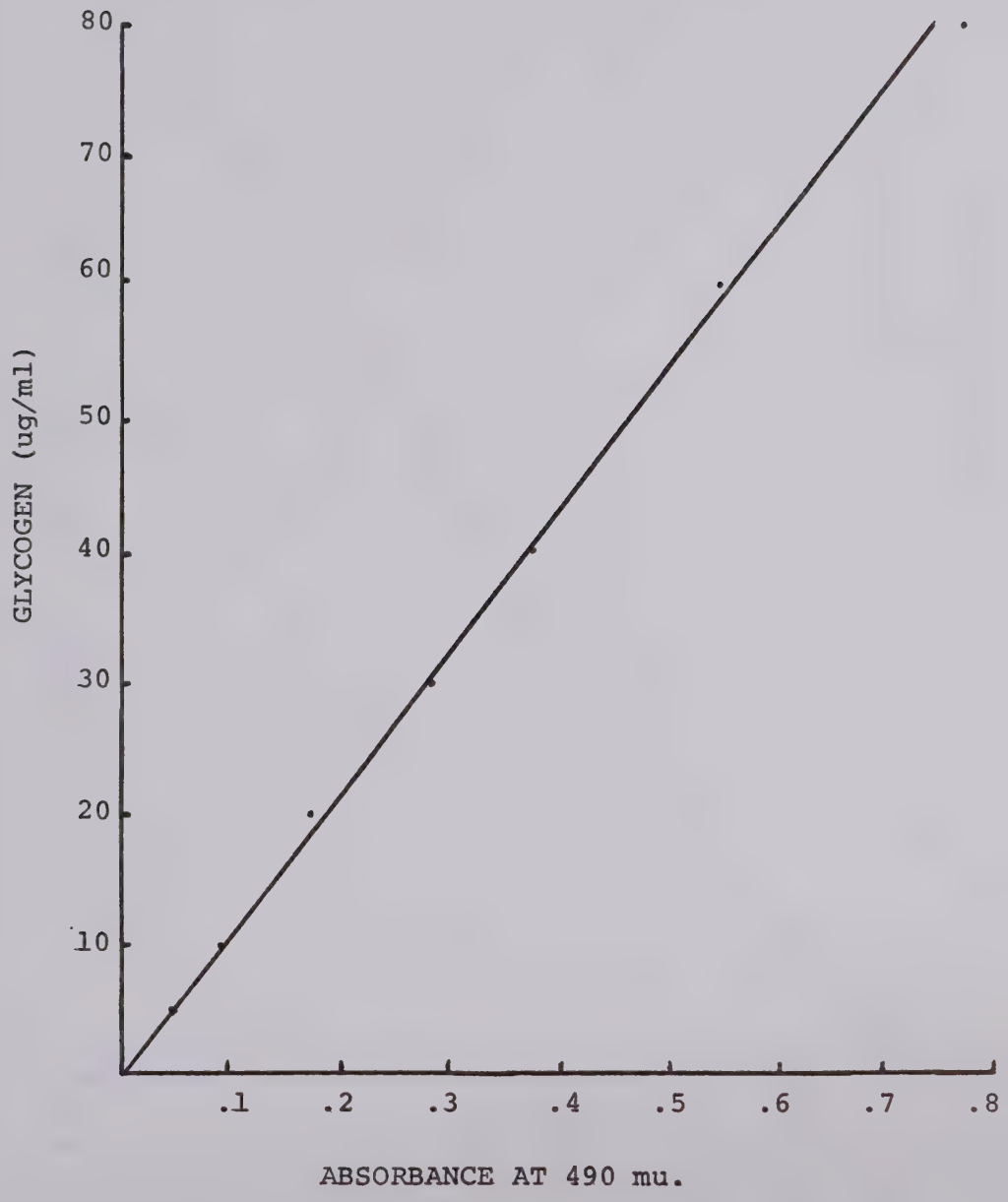


FIGURE 17

STANDARD CURVE FOR LACTIC ACID

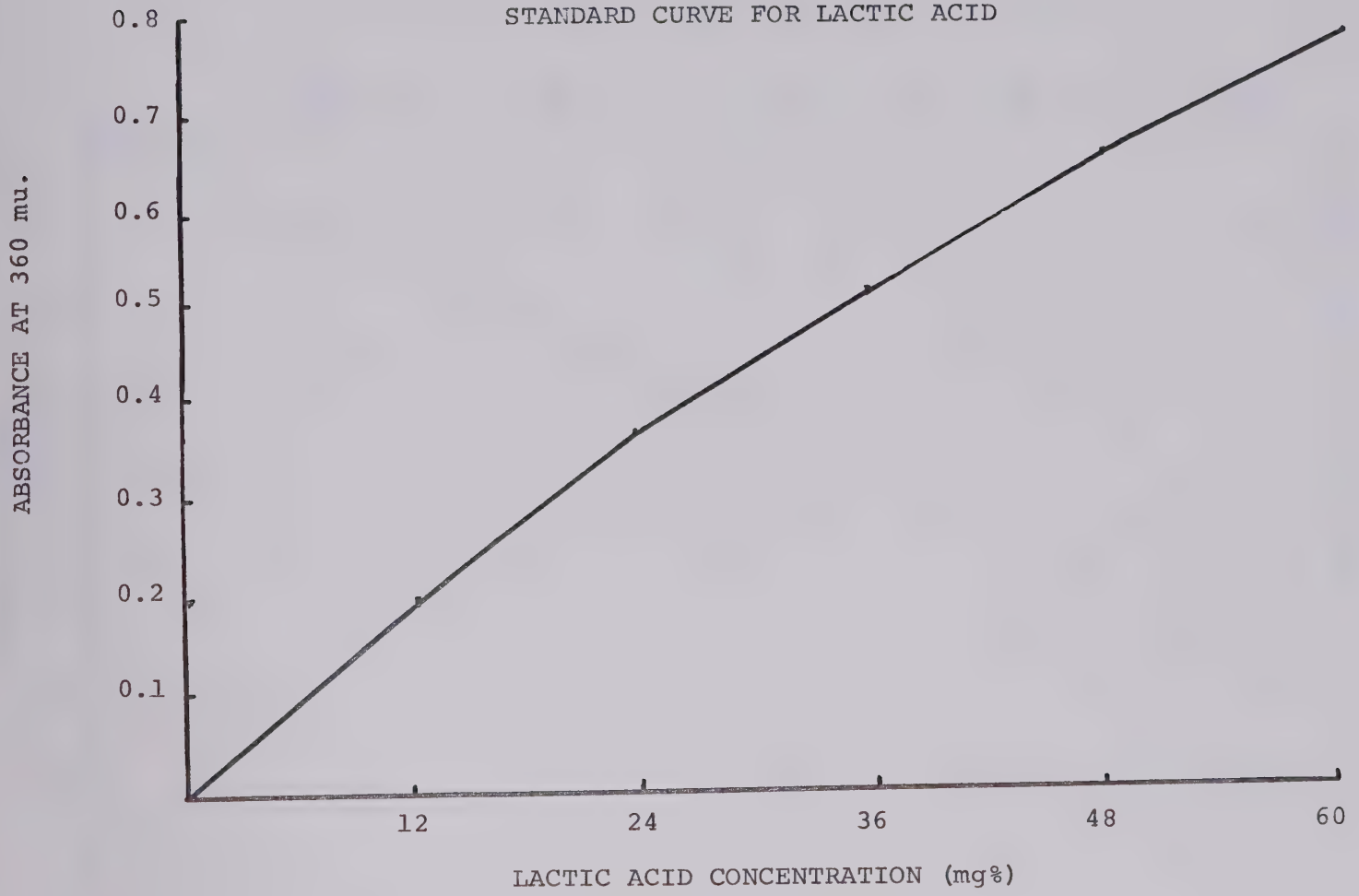
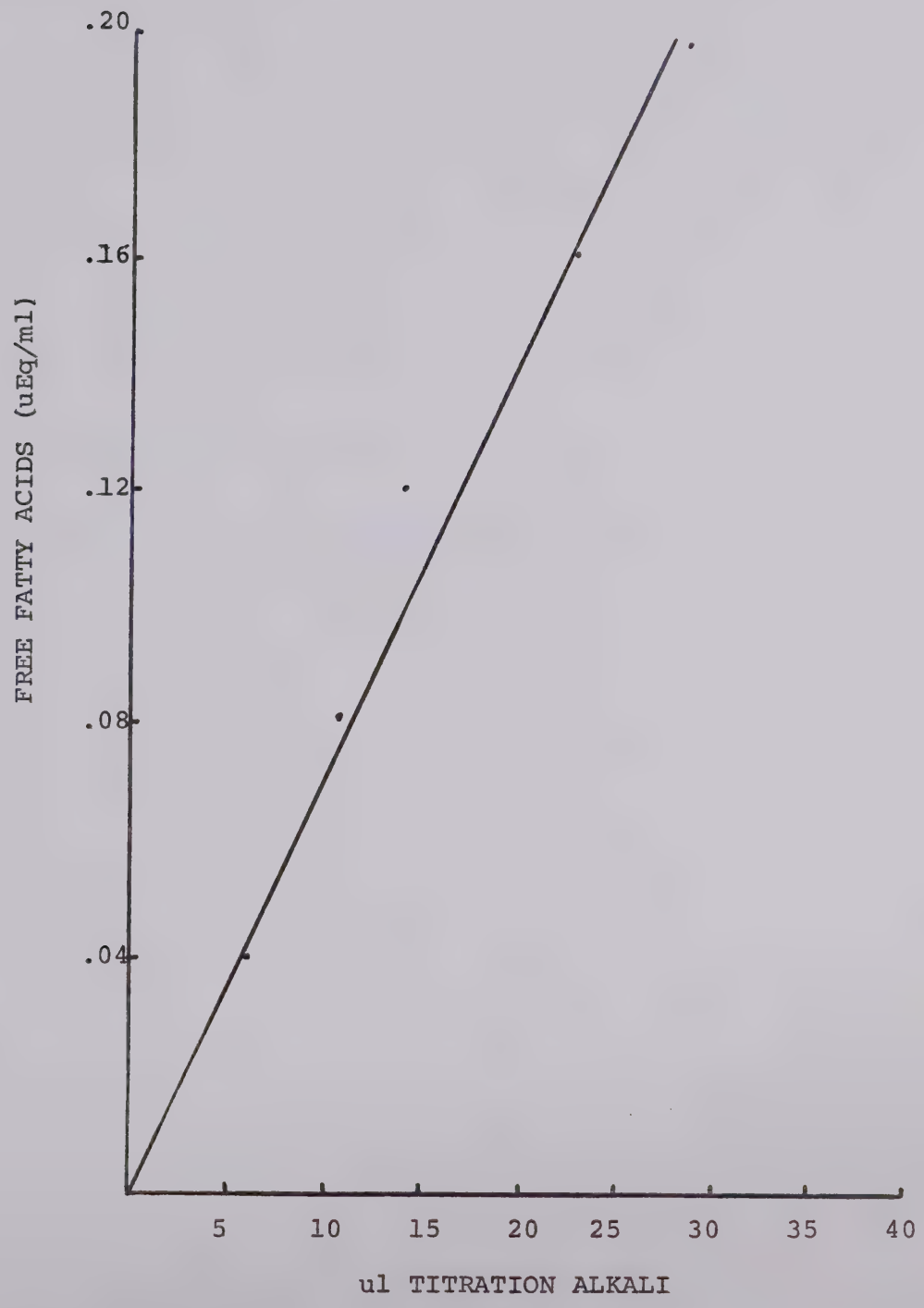


FIGURE 18

STANDARD CURVE FOR FREE FATTY ACIDS



APPENDIX B

TABLE OF MEANS

TABLE 16

MEANS FOR TOTAL BODY WEIGHT
IN Gms

GROUP	TREATMENT NG	ND	CG	CD
NON TRAINED	478.00*	419.50	390.75	357.20
	±25.88	±11.44	±8.13	±25.21
TRAINED REST	451.50	356.60	318.25	367.80
	±22.56	±13.07	±13.21	±8.98
TRAINED FATIGUE	315.00	429.25	343.50	396.00
	±3.87	±15.27	±6.64	±9.59

* means ± standard error of the mean

TREATMENTS FOR ALL TABLES IN APPENDIX B
NG - non castration, non anabolic steroid
ND - non castration, anabolic steroid
CG - castration, non anabolic steroid
CD - castration, anabolic steroid

TABLE 17

MEANS FOR LEFT TESTICLE WEIGHTS
IN Mg

GROUP	TREATMENT NG	ND
NON TRAINED	1553.6*	1566.6
	±80.2	±56.9
TRAINED REST	1584.6	1513.8
	±40.8	±225.0
TRAINED FATIGUE	1524.3	1506.3
	±50.6	±123.9

* means ± standard error of the mean

TABLE 18

MEANS OF RIGHT TESTICLE WEIGHTS
IN Mg

GROUP	TREATMENT NG	ND
NON TRAINED	1514.8*	1551.9
	±127.6	±54.0
TRAINED REST	1597.2	1709.8
	±44.1	±84.1
TRAINED FATIGUE	1529.8	1490.1
	#36.8	±119.9

* means ± standard error of the mean

TABLE 19

MEANS FOR LIVER WEIGHTS
IN Gms

GROUP	NG	TREATMENT ND	CG	CD
NON TRAINED	17.65*	14.32	13.40	10.44
	±1.30	±0.70	±0.58	±0.90
TRAINED REST	17.05	13.30	10.60	11.36
	±0.97	±0.85	±0.92	±0.28
TRAINED FATIGUE	8.75	10.92	9.10	10.12
	±0.19	±0.64	±0.31	±0.39

* means ± standard error of the mean

TABLE 20

MEANS FOR SPLEEN WEIGHTS
IN Mg

GROUP	NG	TREATMENT ND	CG	CD
NON TRAINED	654.25*	624.57	612.80	608.08
	±8.04	±31.91	±28.46	±81.12
TRAINED REST	717.30	593.28	529.05	611.56
	±39.23	±36.65	±50.74	±43.11
TRAINED FATIGUE	483.05	592.75	454.78	565.20
	±3.27	±55.42	±40.67	±29.75

* means ± standard error of the mean

TABLE 21

MEANS FOR HEART WEIGHTS
IN Mg

GROUP	NG	TREATMENT ND	CG	CD
NON TRAINED	1075.9*	979.2	916.3	842.6
	±65.5	±36.8	±3.9	±44.1
TRAINED REST	1032.4	1065.4	785.2	969.7
	±30.9	±37.2	±18.1	±39.7
TRAINED FATIGUE	903.8	1074.5	877.9	995.0
	±5.0	±35.9	±70.1	±19.1

* means ± standard error of the mean

TABLE 22

MEANS FOR ADRENAL WEIGHTS
IN Mg

GROUP	NG	TREATMENT ND	CG	CD
NON TRAINED	52.50*	45.07	52.95	37.36
	±1.26	±3.74	±5.89	±4.20
TRAINED REST	48.60	49.72	55.75	54.44
	±2.26	±2.29	±3.19	±3.83
TRAINED FATIGUE	72.08	47.30	56.50	45.88
	±1.52	±2.65	±4.65	±4.74

* means ± standard error of the mean

TABLE 23

MEANS FOR GASTROCNEMIUS MUSCLE GLYCOGEN
IN Mg/G OF WET TISSUE

GROUP	NG	TREATMENT ND	CG	CD
NON TRAINED	3.477*	1.850	2.585	1.348
	±0.319	±0.236	±0.094	±0.118
TRAINED REST	3.532	2.076	3.270	1.576
	±0.477	±0.215	±0.235	±0.212
TRAINED FATIGUE	1.047	1.030	1.262	0.550
	±0.019	±0.141	±0.363	±0.080

* means ± standard error of the mean

TABLE 24

MEANS FOR BICEPS BRACHII MUSCLE GLYCOGEN
IN Mg/G OF WET TISSUE

GROUP	TREATMENT			
	NG	ND	CG	CD
NON TRAINED	2.457*	1.782	2.642	1.806
	±0.120	±0.265	±0.199	±0.346
TRAINED REST	2.097	1.130	2.060	1.670
	±0.214	±0.925	±0.344	±0.172
TRAINED FATIGUE	0.480	1.272	0.767	0.732
	±0.016	±0.300	±0.222	±0.144

* means ± standard error of the mean

TABLE 25

MEANS FOR LIVER GLYCOGEN
IN Mg/G OF WET TISSUE

GROUP	TREATMENT	
	ND	CD
NON TRAINED	29.632*	41.036
	±11.785	±14.947
TRAINED REST	7.174	23.960
	±3.955	±4.578
TRAINED FATIGUE	1.970	0.736
	±0.833	±0.111

* means ± standard error of the mean

TABLE 26

MEANS FOR HEART MUSCLE GLYCOGEN
IN Mg/G OF WET TISSUE

GROUP	NG	TREATMENT ND	CG	CD
NON TRAINED	1.412*	1.226	1.247	0.930
	±0.154	±0.178	±0.129	±0.129
TRAINED REST	1.312	1.714	1.422	1.000
	±0.122	±0.137	±0.301	±0.143
TRAINED FATIGUE	1.270	1.880	1.485	0.976
	±0.025	±0.241	±0.367	±0.170

* means ± standard error of the mean

TABLE 27

MEANS FOR BLOOD GLUCOSE
IN Mg%

GROUP	NG	TREATMENT ND	CG	CD
NON TRAINED	117.75*	118.83	123.50	120.20
	±5.71	±6.50	±3.52	±3.92
TRAINED REST	117.50	82.20	128.00	123.00
	±5.90	±9.21	±7.27	±10.89
TRAINED FATIGUE	81.00	103.50	92.75	54.80
	±0.82	±18.67	±9.68	±13.76

* means ± standard error of the mean

TABLE 28

MEANS FOR BLOOD LACTATE
IN Mg%

GROUP	NG	TREATMENT ND	CG	CD
NON TRAINED	22.50*	29.50	32.75	46.00
	±2.72	±3.49	±1.93	±12.37
TRAINED REST	21.75	10.70	29.00	18.60
	±2.87	±0.94	±3.16	±3.84
TRAINED FATIGUE	27.00	20.50	60.25	69.00
	±1.96	±2.72	±3.57	±6.91

* means ± standard error of the mean

TABLE 29

MEANS FOR PLASMA FREE FATTY ACIDS
IN uEq/ml

GROUP	NG	TREATMENT ND	CG	CD
NON TRAINED	0.335*	0.388	0.350	0.395
	±0.013	±0.016	±0.024	±0.018
TRAINED REST	0.442	0.499	0.295	0.280
	±0.015	±0.017	±0.039	±0.049
TRAINED FATIGUE	0.667	0.543	0.577	0.487
	±0.055	±0.049	±0.088	±0.070

* means ± standard error of the mean

TABLE 30

MEANS FOR FREE FATTY ACID RELEASE
IN uEq/gm

GROUP	TREATMENT			
	NG	ND	CG	CD
NON TRAINED	1.68*	1.11	1.45	1.30
	±0.53	±0.21	±0.39	±0.25
TRAINED REST	1.90	1.79	2.15	1.44
	±0.23	±0.36	±0.38	±0.39
TRAINED FATIGUE	8.54	3.38	7.56	3.31
	±0.24	±0.56	±1.16	±0.35

* means ± standard error of the mean

APPENDIX C

'NEWMAN-KEULS'

MULTIPLE COMPARISON BETWEEN ORDERED MEANS

TABLE 31

TOTAL BODY WEIGHT (GRAMS)

		ONGR	ENGR	ENDF	ONDR	ECDF	OCGR	ECDR	OCDR	ENDR	ECGF	ECGR	ENGF
	MEAN	478.00	451.50	429.25	419.50	396.00	390.75	367.80	357.60	356.60	343.50	318.25	315.00
ONGR	478.00	---	26.50	48.75	58.50	82.00*	**87.25***	110.20*	120.80*	121.40*	134.50*	159.75*	163.00*
ENGR	451.50		---	22.25	32.00	55.50	60.75	83.70*	94.30*	94.90*	108.00*	133.25*	136.50*
ENDF	429.50			---	9.75	33.25	38.50	61.45	72.05**	72.65**	85.75*	111.00*	114.25*
ONDR	419.50				---	23.50	28.75	51.70	62.30	62.90	76.00**	101.25*	104.50*
ECDF	396.00					---	5.25	28.20	38.80	39.40	52.50	77.75**	81.00**
OCGR	390.75						---	22.95	33.55	34.15	47.25	72.50**	75.75**
ECDR	367.80							---	10.60	11.20	24.30	48.55	52.80
OCDR	357.60								---	0.60	13.70	38.95	42.20
ENDR	356.60									---	13.10	38.35	41.60
ECGF	343.50										---	25.25	28.50
ECGR	318.25											---	3.25

LEGEND FOR ALL TABLES IN APPENDIX C

E - exercise
C - castration
D - anabolic steroid
R - rest
* - P<0.01
O - non exercise
N - non castration
G - non anabolic steroid
F - fatigue
** - P<0.05

TABLE 32

LEFT TESTICLE (MILLIGRAMS)

		ENGR	ONDR	ONGR	ENGF	ENDR	ENDF
	MEAN	1584.550	1566.600	1553.599	1524.299	1513.839	1506.249
ENGR	1584.550	---	17.950	30.951	60.251	70.711	78.301
ONDR	1566.600		---	13.001	42.301	52.761	60.351
ONGR	1553.599			---	29.300	39.760	47.350
ENGF	1524.299				---	10.460	18.050
ENDR	1513.839					---	7.590

TABLE 33

RIGHT TESTICLE (MILLIGRAMS)

		ENDR	ENGR	ONDR	ENGF	ONGR	ENDF
	MEAN	1709.838	1597.149	1551.899	1529.824	1514.749	1490.099
ENDR	1709.838	---	112.689	157.939	180.014	195.089	219.740
ENGR	1597.149		---	45.250	67.325	82.400	107.051
ONDR	1551.899			---	22.075	37.150	61.800
ENGF	1529.824				---	15.075	39.726
ONGR	1514.749					---	24.650

TABLE 34

LIVER WEIGHTS (GRAMS)

		ONGR	ENGR	ONDR	OCGR	ENDR	ECDR	ENDF	ECGR	OCDR	ECDF	ECGF	ENGF
	MEAN	17.65	17.05	14.32	13.40	13.30	11.36	10.92	10.60	10.44	10.12	9.10	8.75
ONGR	17.65	---	0.60	3.33*	4.25*	4.35*	6.29*	6.73*	7.05*	7.21*	7.53*	8.55*	8.90*
ENGR	17.05		---	2.73*	3.65*	3.75*	5.69*	6.13*	6.45*	6.61*	6.93*	7.95*	8.30*
ONDR	14.32			---	0.92	1.02	2.96**	3.40*	3.72*	3.88*	4.20*	5.22*	5.57*
OCGR	13.40				---	0.10	2.04	2.48	2.80**	2.96**	3.28**	4.30*	4.65*
ENDR	13.30					---	1.94**	2.38**	2.70**	2.86**	3.18**	4.20*	4.55*
ECDR	11.36						---	0.44	0.76	0.92	1.24	2.26	2.61
ENDF	10.92							---	0.32	0.48	0.80	1.82	2.17
ECGR	10.60								---	0.16	0.48	1.50	1.85
OCDR	10.44									---	0.32	1.34	1.69
ECDF	10.12										---	1.02	1.37
ECGF	9.10											---	0.35

TABLE 35

SPLEEN WEIGHTS (MILLIGRAMS)

		ENGR	ONGR	ENDR	ONDR	OCGR	OCDR	ECDR	ENDF	ECDF	ECGR	ENGF	ECGF
	MEAN	717.30	654.25	640.84	624.57	612.80	608.08	593.28	592.75	565.20	529.05	483.05	454.78
ENGR	717.30	---	63.05	76.46	92.73	104.50	109.22	124.02	124.55	152.10	188.25**	234.25*	262.53*
ONGR	654.25		---	13.41	29.68	41.45	46.17	60.97	61.50	89.05	125.20	171.20	199.48**
ENDR	640.84			---	16.27	28.04	32.76	47.56	48.09	75.64	111.79	157.79	186.07**
ONDR	624.57				---	11.77	16.48	31.29	31.82	59.37	95.52	141.52	169.79
OCGR	612.80					---	4.72	19.52	20.05	47.60	83.75	129.75	158.03
OCDR	608.08						---	14.80	15.33	42.88	79.03	125.03	153.31
ECDR	593.28							---	0.53	28.08	64.23	110.23	138.51
ENDF	592.75								---	27.55	63.70	109.70	137.98
ECDF	565.20									---	36.15	82.15	110.43
ECGR	529.05										---	46.00	74.28
ENGF	483.05											---	28.28

TABLE 36

HEART WEIGHTS (MILLIGRAMS)

		ONGR	ENDF	ENDR	ENGR	ECDF	ONDR	ECDR	OCGR	ENGF	ECGF	OCDR	ECGR
	MEAN	1075.9	1074.5	1065.4	1032.4	995.0	979.2	969.7	916.3	903.8	877.9	842.6	875.2
ONGR	1075.9	---	1.4	10.5	43.5	80.9	86.7	106.2	159.6	172.1	198.0**	233.3*	290.7*
ENDF	1074.5		---	9.1	42.0	79.5	95.3	104.8	158.2	170.7	196.6**	231.8*	289.3*
ENDR	1065.4			---	32.0	70.4	86.2	95.7	149.1	161.7	187.6**	222.8*	280.2*
ENGR	1032.4				---	37.4	53.3	62.8	116.1	128.7	154.6	189.9**	247.3*
ECDF	995.0					---	15.8	25.3	78.7	91.3	117.2	152.4	209.8*
ONDR	979.2						---	9.5	62.9	75.4	101.3	136.5	194.0**
ECDR	969.7							---	53.4	65.9	91.8	127.0	184.5**
OCGR	916.3								---	12.6	38.5	73.7	131.1
ENGF	903.8									---	25.9	61.1	118.6
ECGF	877.8										---	35.2	92.7
OCDR	842.6											---	57.5

TABLE 37

ADRENAL WEIGHTS (MILLIGRAMS)

		ENGF	ECGF	ECGR	ECDR	OCGR	ONGR	ENDR	ENGR	ENDF	ECDF	ONDR	OCDR
	MEAN	72.08	56.50	55.75	54.44	52.95	52.50	49.72	48.60	47.30	45.88	45.07	37.36
ENGF	72.08	---	15.58*	16.33*	17.64*	19.13*	19.58*	22.36*	23.48*	24.78*	26.20*	29.01*	34.72*
ECGF	56.50		---	0.75	2.06	3.55	4.00	6.78	7.90	9.20	10.62	11.43	19.14*
ECGR	55.75			---	1.31	2.80	3.25	6.03	7.15	8.45	9.87	10.68	18.39*
ECDR	54.44				---	1.49	1.94	4.72	5.84	7.14	8.56	9.37	17.08**
OCGR	52.95					---	0.45	3.23	4.35	5.65	7.07	7.88	15.59**
ONGR	52.50						---	2.78	3.90	5.20	6.62	7.43	15.14**
ENDR	49.72							---	1.12	2.42	3.84	4.65	12.36
ENGR	48.60								---	1.30	2.72	3.53	11.24
ENDF	47.30									---	1.42	2.23	9.94
ECDF	45.88										---	0.81	8.52
ONDR	45.07											---	7.71

TABLE 38

GASTROCNEMIUS MUSCLE GLYCOGEN CONTENT (MILLIGRAMS PER GRAM WET MUSCLE TISSUE)

		ENGR	ONGR	ECGR	OCGR	ENDR	ONDR	ECDR	OCDR	ECGF	ENFG	ENDF	ECDF
	MEAN	3.532	3.477	3.270	2.585	2.076	1.850	1.576	1.348	1.262	1.047	1.030	0.550
ENGR	3.532	---	0.055	0.262	0.948	1.457*	1.682*	1.957*	2.184*	2.270*	2.485*	2.503*	2.982*
ONGR	3.477		---	0.207	0.892	1.401*	1.627*	1.901*	2.129*	2.215*	2.430*	2.448*	2.927*
ECGR	3.270			---	0.685	1.194**	1.420*	1.694*	1.922*	2.008*	2.223*	2.240*	2.720*
OCGR	2.585				---	0.509	0.735	1.009	1.237**	1.323**	1.538*	1.555*	2.035*
ENDR	2.076					---	0.226	0.500	0.728	0.814	1.029	1.046	1.526*
ONDR	1.850						---	0.274	0.502	0.588	0.803	0.820	1.300**
ECDR	1.576							---	0.228	0.314	0.529	0.546	1.026
OCDR	1.348								---	0.086	0.301	0.318	0.798
ECGF	1.262									---	0.215	0.233	0.712
ENGF	1.047										---	0.017	0.497
ENDF	1.030											---	0.480

TABLE 39

BICEP BRACHII MUSCLE GLYCOGEN CONTENT (MILLIGRAMS PER GRAM WET MUSCLE TISSUE)

		OCGR	ONGR	ENGR	ECGR	OCDR	ONDR	ECDR	ENDF	ENDR	ECGF	ECDF	ENGF
	MEAN	2.642	2.457	2.097	2.060	1.806	1.782	1.670	1.272	1.130	0.767	0.732	0.480
OCGR	2.642	---	0.185	0.545	0.582	0.836	0.861	0.972**	1.370*	1.512*	1.875*	1.910*	2.162*
ONGR	2.457		---	0.360	0.397	0.651	0.676	0.787	1.185*	1.327*	1.690*	1.725*	1.977*
ENGR	2.097			---	0.037	0.291	0.316	0.427	0.825	0.967**	1.330*	1.365*	1.617*
ECGR	2.060				---	0.254	0.278	0.390	0.788	0.930**	1.292*	1.328*	1.580*
OCDR	1.806					---	0.024	0.136	0.534	0.676	1.038**	1.074*	1.326*
ONDR	1.782						---	0.112	0.509	0.652	1.014**	1.050*	1.302*
ECDR	1.670							---	0.398	0.540	0.902**	0.938**	1.190*
ENDF	1.272								---	0.142	0.505	0.540	0.792
ENDR	1.130									---	0.362	0.398	0.650
ECGF	0.767										---	0.036	0.287
ECDF	0.732											---	0.252

TABLE 40

LIVER GLYCOGEN (MILLIGRAMS PER GRAM WET TISSUE)

		OCDR	ONDR	ECDR	ENDR	ENDF	ECDF
	MEAN	41.036	29.632	23.960	7.174	1.970	0.736
OCDR	41.036	---	11.404	17.076	33.862**	39.066**	40.300**
ONDR	29.632		---	5.672	22.458**	27.662**	28.896**
ECDR	23.960			---	16.786	21.990**	23.224**
ENDR	7.174				---	5.204	6.438
ENDF	1.970					---	1.234

TABLE 41

HEART MUSCLE GLYCOGEN CONTENT (MILLIGRAMS PER GRAM WET MUSCLE TISSUE)

		ENDF	ENGR	ECGF	ECGR	ONGR	ENGR	ENGF	OCGR	ONDR	ECDR	ECDF	OCDR
	MEAN	1.880	1.714	1.485	1.422	1.412	1.312	1.270	1.247	1.227	1.000	0.976	0.930
ENDF	1.880	---	0.166	0.395	0.458	0.467	0.568	0.610	0.632	0.653	0.880	0.904	0.950**
ENGR	1.714		---	0.229	0.292	0.301	0.401	0.444	0.466	0.487	0.714	0.738	0.784
ECGF	1.485			---	0.063	0.073	0.173	0.215	0.238	0.258	0.485	0.509	0.555
ECGR	1.422				---	0.010	0.110	0.153	0.175	0.196	0.422	0.446	0.492
ONGR	1.412					---	0.100	0.143	0.165	0.186	0.412	0.437	0.482
ENGR	1.312						---	0.043	0.065	0.086	0.312	0.336	0.382
ENGF	1.270							---	0.022	0.043	0.270	0.294	0.340
OCGR	1.247								---	0.021	0.247	0.272	0.317
ONDR	1.227									---	0.227	0.251	0.297
ECDR	1.000										---	0.024	0.070
ECDF	0.976											---	0.046

TABLE 42

BLOOD GLUCOSE (MILLIGRAMS PER CENT)

		ECGR	OCGR	ECDR	OCDR	ONDR	ONGR	ENGR	ENDF	ECGF	ENDR	ENGF	ECDF
	MEAN	128.00	123.50	123.00	120.20	118.83	117.75	117.50	103.5	92.75	82.20	81.00	54.80
ECGR	128.00	---	4.50	5.00	7.80	9.17	10.25	10.50	24.50	35.25	45.80**	47.00**	73.20*
OCGR	123.50		---	0.50	3.30	4.67	5.75	6.00	20.00	30.75	41.30	42.50	68.70*
ECDR	123.00			---	2.80	4.17	5.25	5.50	19.50	30.25	40.80	42.00	68.20*
OCDR	120.20				---	1.37	2.45	2.70	16.70	27.45	38.00	39.20	65.40*
ONDR	118.83					---	1.08	1.33	15.33	26.08	36.63	37.83	64.03*
ONGR	117.75						---	0.25	14.25	25.00	35.55	36.75	62.95*
ENGR	117.50							---	14.00	24.75	35.30	36.50	62.70*
ENDF	103.50								---	10.75	21.30	22.50	48.70*
ECGF	92.75									---	10.55	11.75	37.95**
ENDR	82.20										---	1.20	27.40
ENGF	81.00											---	26.20

TABLE 43

BLOOD LACTATE (MILLIGRAMS PER CENT)

		ECDF	ECGF	OCDR	OCGR	ONDR	ECGR	ENGF	ONGR	ENGR	ENDF	ECDR	ENDR
	MEAN	69.00	60.25	46.00	32.75	29.50	29.00	27.00	22.50	21.75	20.50	18.60	10.70
ECDF	69.00	---	8.75	23.00**	36.25*	39.50*	40.00*	42.00*	46.50*	47.25*	48.50*	50.40*	58.30*
ECGF	60.25		---	14.25	27.50**	30.75**	31.25*	33.25*	37.75*	38.50*	39.75*	41.65*	49.55*
OCDR	46.00			---	13.25	16.50	17.00	19.00	23.50	24.25	25.50	27.40	35.30**
OCGR	32.75				---	3.25	3.75	5.75	10.25	11.00	12.25	14.15	22.05
ONDR	29.50					---	0.500	2.50	7.00	7.75	9.00	10.90	18.80
ECGR	29.00						---	2.00	6.50	7.25	8.50	10.40	18.30
ENGF	27.00							---	4.50	5.25	6.50	8.40	16.30
ONGR	22.50								---	0.75	2.00	3.90	11.80
ENGR	21.75									---	1.25	3.15	11.05
ENDF	20.50										---	1.90	9.80
ECDR	18.60											---	7.90

TABLE 44

PLASMA FREE FATTY ACID (MICRO-EQUIVALENTS PER MILLILITRE)

		ENGF	ECGF	ENDF	ENDR	ECDF	ENGR	OCDR	ONDR	OCGR	ONGR	ECGR	ECDR
	MEAN	0.667	0.577	0.543	0.499	0.487	0.442	0.395	0.388	0.350	0.335	0.295	0.280
ENGF	p.667	---	0.090	0.124	0.169	0.181	0.225	0.272*	0.279*	0.317*	0.332)	0.372*	0.387*
ECGF	0.577		---	0.034	0.079	0.091	0.135	0.182	0.189	0.227**	0.242**	0.282*	0.297*
ENDF	0.543			---	0.044	0.056	0.100	0.148	0.155	0.193	0.208	0.248**	0.263*
ENDR	0.499				---	0.012	0.056	0.103	0.111	0.149	0.164	0.204	0.218**
ECDF	0.487					---	0.044	0.091	0.099	0.137	0.152	0.192	0.207
ENGR	0.442						---	0.047	0.055	0.092	0.108	0.148	0.162
OCDR	0.395							---	0.007	0.045	0.060	0.100	0.115
ONDR	0.388								---	0.038	0.053	0.093	0.108
OCGR	0.350									---	0.015	0.055	0.070
ONGR	0.335										---	0.040	0.055
ECGR	0.295											---	0.015

TABLE 45

FREE FATTY ACID RELEASE (MICRO-EQUIVALENTS PER GRAM)

		ENGF	ECGF	ENDR	ENDF	ECGR	ENGR	ENDF	ONGR	OCGR	ECDR	OCDR	ONDR
	MEAN	8.54	7.56	3.38	3.31	2.15	1.90	1.79	1.68	1.45	1.44	1.30	1.11
LNGF	8.54	---	0.98	5.16*	5.23*	6.39*	6.64*	6.75*	6.86*	7.09*	7.10*	7.24*	7.43*
ECGF	7.56		---	4.18*	4.25*	5.41*	5.66*	5.77*	5.88*	6.11*	6.12*	6.26*	6.45*
ENDR	3.38			---	0.07	1.23	1.48	1.59	1.71	1.93**	1.94	2.08**	2.28**
ECDF	3.31				---	1.16	1.41	1.52	1.64	1.86	1.87	2.01**	2.20**
ECGR	2.15					---	0.25	0.36	0.47	0.70	0.71	0.85	1.04
ENGR	1.90						---	0.11	0.23	0.46	0.47	0.61	0.80
ENDF	1.79							---	0.12	0.35	0.36	0.50	0.69
ONGR	1.68								---	0.23	0.24	0.38	0.57
OCGR	1.45									---	0.01	0.15	0.34
ECDR	1.44										---	0.14	0.33
OCDR	1.30											---	0.19

APPENDIX D

RAW DATA

TABLE 46

TOTAL BODY WEIGHTS FOR EXPERIMENTAL ANIMALS

No.	NON-TRAINED	No.	TRAINED REST	No.	TRAINED FATIGUE
			NORMAL RATS		
81	512	73	500	77	304
82	524	74	425	78	316
83	466	75	403	79	322
84	410	76	478	80	318
			ANABOLIC STEROID TREATED RATS		
9	443	26	374	6	433
36	456	27	384	7	448
37	409	28	372	8	451
38	378	29	339	10	385
39	425	30	314		
40	406				
			CASTRATED RATS		
69	385	61	282	65	356
70	415	62	326	66	328
71	381	63	345	67	337
72	382	64	320	68	353
			CASTRATED AND ANABOLIC STEROID TREATED RATS		
31	414	21	379	1	426
32	364	22	343	2	397
33	268	23	375	3	404
34	394	24	351	4	384
35	346	25	391	5	369

TABLE 47

LEFT TESTICLE WEIGHTS FOR EXPERIMENTAL ANIMALS

No.	NON-TRAINED	No.	TRAINED REST	No.	TRAINED FATIGUE
			NORMAL RATS		
81	1484.8	73	1637.2	77	1400.6
82	1741.4	74	1515.0	78	1486.3
83	1616.8	75	1515.0	79	1626.9
84	1371.4	76	1671.0	80	1583.4
			ANABOLIC STEROID TREATED RATS		
9	1463.0	26	1577.4	6	1354.2
36	1663.0	27	1789.8	7	1252.2
37	1408.0	28	2026.0	8	1625.0
38	1670.2	29	1479.8	10	1793.6
39	1738.2	30	696.2		
40	1457.2				

TABLE 48

RIGHT TESTICLE WEIGHTS FOR EXPERIMENTAL ANIMALS

No.	NON-TRAINED	No.	TRAINED REST	No.	TRAINED FATIGUE
			NORMAL RATS		
81	1567.8	73	1613.2	77	1423.6
82	1712.8	74	1573.2	78	1623.8
83	1636.0	75	1495.0	79	1529.3
84	1142.4	76	1707.2	80	1542.6
			ANABOLIC STEROID TREATED RATS		
9	1490.6	26	1625.2	6	1344.2
36	1472.0	27	1789.8	7	1258.6
37	1639.0	28	1994.0	8	1564.0
38	1370.0	29	1506.4	10	1793.6
39	1604.0	30	1633.8		
40	1735.8				

TABLE 49

LIVER WEIGHTS FOR EXPERIMENTAL ANIMALS

No.	NON-TRAINED	No.	TRAINED REST	No.	TRAINED FATIGUE
			NORMAL RATS		
81	19.6	73	18.8	77	8.52
82	19.4	74	16.4	78	9.31
83	17.6	75	14.6	79	8.73
84	14.0	76	18.4	80	8.46
			ANABOLIC STEROID TREATED RATS		
9	14.2	26	13.6	6	11.4
36	16.0	27	14.4	7	13.2
37	13.0	28	14.4	8	9.8
38	12.1	29	10.8	10	9.8
39	16.6	30	13.9		
40	14.0				
			CASTRATED RATS		
69	12.2	61	8.4	65	9.6
70	15.0	62	11.8	66	8.2
71	13.2	63	12.4	67	9.2
72	13.2	64	9.8	68	9.4
			CASTRATED AND ANABOLIC STEROID TREATED RATS		
31	11.6	21	11.0	1	11.2
32	11.6	22	11.4	2	10.8
33	7.2	23	10.8	3	9.8
34	12.0	24	11.2	4	9.0
35	9.8	25	12.4	5	9.8

TABLE 50

SPLEEN WEIGHTS FOR EXPERIMENTAL ANIMALS

No.	NON-TRAINED	No.	TRAINED REST	No.	TRAINED FATIGUE
			NORMAL RATS		
81	665.2	73	817.0	77	484.1
82	664.2	74	652.8	78	480.2
83	630.8	75	656.4	79	476.3
84	656.8	76	743.0	80	491.6
			ANABOLIC STEROID TREATED RATS		
9	702.4	26	504.0	6	526.0
36	660.0	27	722.6	7	617.6
37	547.0	28	713.6	8	736.0
38	515.0	29	646.2	10	489.4
39	696.8	30	617.8		
40	626.2				
			CASTRATED RATS		
69	545.0	61	432.4	65	570.0
70	682.0	62	670.6	66	437.3
71	599.0	63	490.6	67	378.6
72	625.0	64	522.6	68	433.2
			CASTRATED AND ANABOLIC STEROID TREATED RATS		
31	902.8	21	591.2	1	654.4
32	615.4	22	562.2	2	609.8
33	446.4	23	582.0	3	491.0
34	603.8	24	726.8	4	520.0
35	472.0	25	504.2	5	550.8

TABLE 51

HEART WEIGHTS FOR EXPERIMENTAL ANIMALS

No.	NON-TRAINED	No.	TRAINED REST	No.	TRAINED FATIGUE
			NORMAL RATS		
81	1160.6	73	1101.4	77	890.2
82	1207.2	74	960.4	78	902.4
83	1011.0	75	1006.5	79	910.6
84	924.8	76	1061.4	80	911.8
			ANABOLIC STEROID TREATED RATS		
9	1087.8	26	1071.8	6	1012.2
36	1025.0	27	1204.6	7	1177.8
37	985.0	28	1037.0	8	1050.2
38	819.0	29	1023.6	10	1057.6
39	1002.2	30	990.0		
40	956.0				
			CASTRATED RATS		
69	914.8	61	744.4	65	1028.4
70	927.6	62	832.4	66	793.6
71	910.2	63	779.1	67	728.6
72	912.6	64	784.8	68	960.8
			CASTRATED AND ANABOLIC STEROID TREATED RATS		
31	984.4	21	1034.4	1	1026.4
32	824.8	22	918.2	2	1026.6
33	714.0	23	847.6	3	975.0
34	876.0	24	980.0	4	929.0
35	814.0	25	1068.2		

TABLE 52

ADRENAL WEIGHTS FOR EXPERIMENTAL ANIMALS

No.	NON-TRAINED	No.	TRAINED REST	No.	TRAINED FATIGUE
			NORMAL RATS		
81	50.8	73	51.4	77	72.2
82	56.2	74	43.0	78	76.3
83	51.0	75	47.0	79	69.4
84	52.0	76	53.0	80	70.4
			ANABOLIC STEROID TREATED RATS		
9	46.4	26	45.6	6	44.0
36	63.0	27	43.2	7	42.0
37	39.0	28	51.6	8	53.6
38	40.0	29	55.4	10	49.6
39	41.8	30	52.8		
40	40.2				
			CASTRATED RATS		
69	47.2	61	54.4	65	64.6
70	70.6	62	58.4	66	60.4
71	46.6	63	62.6	67	43.2
72	47.4	64	47.6	68	57.8
			CASTRATED AND ANABOLIC STEROID TREATED RATS		
31	44.6	21	67.6	1	62.4
32	34.0	22	48.4	2	34.0
33	26.0	23	46.4	3	46.0
34	49.2	24	58.0	4	47.0
35	33.0	25	51.8	5	40.0

TABLE 53

GASTROCNEMIUS MUSCLE GLYCOGEN FOR EXPERIMENTAL ANIMALS

No.	NON-TRAINED	No.	TRAINED REST	No.	TRAINED FATIGUE
			NORMAL RATS		
81	3.63	73	4.06	77	1.03
82	4.22	74	3.28	78	1.05
83	3.38	75	2.31	79	1.01
84	2.68	76	4.48	80	1.10
			ANABOLIC STEROID TREATED RATS		
9	1.61	26	2.04	6	1.43
36	0.93	27	2.00	7	0.93
37	2.66	28	1.44	8	0.99
38	1.87	29	2.11	10	0.77
39	2.18	30	2.79		
40	1.85				
			CASTRATED RATS		
69	2.82	61	2.88	65	1.63
70	2.56	62	3.42	66	0.97
71	2.60	63	3.87	67	0.40
72	2.36	64	2.91	68	2.05
			CASTRATED AND ANABOLIC STEROID TREATED RATS		
31	1.40	21	0.95	1	0.48
32	1.26	22	1.79	2	0.40
33	1.73	23	1.74	3	0.85
34	1.00	24	1.25	4	0.45
35	1.35	25	2.15	5	0.57

TABLE 54

BICEP BRACHII MUSCLE GLYCOGEN FOR EXPERIMENTAL ANIMALS

No.	NON-TRAINED	No.	TRAINED REST	No.	TRAINED FATIGUE
			NORMAL RATS		
81	2.71	73	2.34	77	0.48
82	2.61	74	1.93	78	0.52
83	2.30	75	2.54	79	0.44
84	2.21	76	1.58	80	0.48
			ANABOLIC STEROID TREATED RATS		
9	1.82	26	1.08	6	1.41
36	1.59	27	1.41	7	2.00
37	1.11	28	1.04	8	1.12
38	1.99	29	0.87	10	0.56
40	1.26				
			CASTRATED RATS		
69	2.43	61	1.15	65	1.16
70	3.24	62	1.93	66	0.15
71	2.45	63	2.71	67	0.76
72	2.45	64	2.45	68	1.00
			CASTRATED AND ANABOLIC STEROID TREATED RATS		
31	1.70	21	1.05	1	0.73
32	1.81	22	2.08	2	0.64
33	0.91	23	1.67	3	0.55
34	1.57	24	1.69	4	0.46
35	3.04	25	1.86	5	1.28

TABLE 55

LIVER GLYCOGEN FOR EXPERIMENTAL ANIMALS

No.	NON-TRAINED	No.	TRAINED REST	No.	TRAINED FATIGUE
			ANABOLIC STEROID TREATED RATS		
9	1.70	26	4.80	6	0.39
36	53.20	27	22.80	7	2.71
37	6.85	28	1.46	8	3.96
38	46.80	29	2.41	10	0.82
39	66.00	30	4.40		
40	3.24				
			CASTRATED AND ANABOLIC STEROID TREATED RATS		
31	74.80	21	16.80	1	0.57
32	56.00	22	34.00	2	0.41
33	7.00	23	28.40	3	0.85
34	64.00	24	30.80	4	1.04
35	3.38	25	9.80	5	0.81

TABLE 56

HEART MUSCLE GLYCOGEN FOR EXPERIMENTAL ANIMALS

No.	NON-TRAINED	No.	TRAINED REST	No.	TRAINED FATIGUE
			NORMAL RATS		
81	1.70	73	1.49	77	1.26
82	1.01	74	0.99	78	1.34
83	1.34	75	1.26	79	1.22
84	1.60	76	1.51	80	1.26
			ANABOLIC STEROID TREATED RATS		
9	0.80	26	1.66	6	2.30
36	1.90	27	1.53	7	1.41
37	0.99	28	1.39	8	2.29
38	0.85	29	1.80	10	1.52
39	1.57	30	2.19		
40					
			CASTRATED RATS		
69	1.34	61	1.69	65	1.06
70	1.03	62	0.64	66	1.64
71	1.57	63	2.05	67	0.79
72	1.05	64	1.31	68	2.45
			CASTRATED AND ANABOLIC STEROID TREATED RATS		
31	1.70	21	0.78	1	1.09
32	1.81	22	1.14	2	0.84
33	0.91	23	0.83	3	0.88
34	1.57	24	1.50	4	0.52
35	3.04	25	0.75	5	1.55

TABLE 57

BLOOD GLUCOSE FOR EXPERIMENTAL ANIMALS

No.	NON-TRAINED	No.	TRAINED REST	No.	TRAINED FATIGUE
			NORMAL RATS		
81	125	73	124	77	81
82	101	74	100	78	79
83	125	75	121	79	83
84	120	76	125	80	81
			ANABOLIC STEROID TREATED RATS		
9	143	26	64	6	138
36	103	27	107	7	124
37	108	28	58	8	99
38	134	29	93	10	53
39	114	30	89		
40	111				
			CASTRATED RATS		
69	122	61	111	65	86
70	125	62	121	66	93
71	115	63	138	67	73
72	132	64	142	68	119
			CASTRATED AND ANABOLIC STEROID TREATED RATS		
31	112	21	108	1	55
32	117	22	162	2	51
33	135	23	132	3	34
34	117	24	106	4	28
35	120	25	107	5	106

TABLE 58

BLOOD LACTATE FOR EXPERIMENTAL ANIMALS

No.	NON-TRAINED	No.	TRAINED REST	No.	TRAINED FATIGUE
			NORMAL RATS		
81	30.0	73	15.0	77	22.0
82	23.0	74	22.0	78	31.0
83	19.0	75	29.0	79	26.0
84	18.0	76	21.0	80	29.0
			ANABOLIC STEROID TREATED RATS		
9	23.0	26	10.0	6	20.0
36	31.0	27	13.0	7	28.0
37	26.0	28	7.5	8	15.0
38	27.0	29	11.0	10	19.0
40	46.0				
			CASTRATED RATS		
69	35.0	61	31.0	65	70.0
70	35.0	62	25.0	66	58.0
71	27.0	63	37.0	67	60.0
72	34.0	64	23.0	68	53.0
			CASTRATED AND ANABOLIC STEROID TREATED RATS		
31	16.0	21	12.0	1	69.0
32	47.0	22	30.0	2	84.0
33	90.0	23	10.0	3	81.0
34	46.0	24	25.0	4	66.0
35	31.0	25	16.0	5	45.0

TABLE 59

PLASMA FREE FATTY ACIDS FOR EXPERIMENTAL ANIMALS

No.	NON-TRAINED	No.	TRAINED REST	No.	TRAINED FATIGUE
			NORMAL RATS		
81	0.320	73	0.450	77	0.550
82	0.310	74	0.470	78	0.630
83	0.370	75	0.450	79	0.810
84	0.340	76	0.400	80	0.680
			ANABOLIC STEROID TREATED RATS		
9	0.345	26	0.552	6	0.672
36	0.417	27	0.480	7	0.540
37	0.396	28	0.468	8	0.435
38	0.450	29	0.525	10	0.525
39	0.360	30	0.468		
40	0.360				
			CASTRATED RATS		
69	0.340	61	0.300	65	0.470
70	0.420	62	0.270	66	0.830
71	0.320	63	0.210	67	0.560
72	0.320	64	0.400	68	0.450
			CASTRATED AND ANABOLIC STEROID TREATED RATS		
31	0.345	21	0.405	1	0.487
32	0.399	22	0.285	2	0.294
33	0.435	23	0.372	3	0.525
34	0.366	24	0.168	4	0.720
35	0.432	25	0.171	5	0.408

TABLE 60

FREE FATTY ACID RELEASE FOR EXPERIMENTAL ANIMALS

No.	NON-TRAINED	No.	TRAINED REST	No.	TRAINED FATIGUE
			NORMAL RATS		
81	0.76	73	1.56	77	9.09
82	2.21	74	1.61	78	8.76
83	2.91	75	2.57	79	7.99
84	0.82	76	1.87	80	8.31
			ANABOLIC STEROID TREATED RATS		
9	1.97	26	2.87	6	1.66
36	1.08	27	5.35	7	1.75
37	1.41	28	1.92	8	2.76
38	0.69	29	3.38	10	1.00
39	0.94	30	3.39		
40	0.55				
			CASTRATED RATS		
69	1.21	61	2.91	65	10.34
70	2.49	62	1.95	66	8.16
71	0.60	63	2.55	67	4.77
72	1.49	64	1.18	68	6.96
			CASTRATED AND ANABOLIC STEROID TREATED RATS		
31	1.58	21	2.51	1	2.74
32	0.54	22	1.38	2	3.50
33	1.07	23	1.42	3	3.51
34	2.05	24	1.76	4	2.41
35	1.24	25	0.12	5	4.39

TABLE 61

ENDURANCE FATIGUE TIME FOR EXPERIMENTAL ANIMALS IN MINUTES

ANABOLIC STEROID TREATED RATS

No.	1.0 mph	1.25 mph	1.5 mph
6	120	25	---
7	106	--	---
8	116	10	---
10	116	20	---

CASTRATED AND ANABOLIC STEROID TREATED RATS

No.	1.0 mph	1.25 mph	1.5 mph
1	123	--	--
2	124	28	1
3	115	30	23
4	115	30	29
5	120	30	6